Journal of the
Indian Medical Association

VOL. XV, No. 9 CALCUTTA JUNE, 1946

VAN SLYKE'S UREA CLEARANCE TEST IN INDIANS IN HEALTH AND DISEASE

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This paper contains a discussion on the urea clearance test as elaborated by the work of Addis and Van Slyke.

EXPERIMENTAL DETAILS

I. Types of cases investigated:

These are divided into two groups:—

(*i*) Healthy persons serving as control.

The healthy persons serving as controls are divided into three sub-groups:—

(*a*) Medical students. Thirty cases are included in this series. Age varies between 20 and 25 years except in five cases where it was between 25 and 30. There are five Muslims and 25 Hindus amongst them. This provided the data pertaining to healthy young men in this part of our country.

(*b*) Students from the High School classes. This includes only ten cases, 2 Muslims and 8 Hindus. Age varies from 14 to 20 years, giving the data for adolescents.

(*c*) Laboratory assistants and Laboratory boys representing wider age limits. Four Muslims and six Hindus are included in this group, a total of ten cases. Age varies from 19 to 42 years.

(*ii*) Diseased persons selected from patients admitted to medical and surgical wards of the Agra Medical College Hospital in order to investigate the renal functional capacity in different types of diseases.

These cases consisted of the following four different types of patients.

(*a*) Nephritics: This group of patients has been fully investigated as regards the condition of their excretory organs, and the stage of the disease shown by them when admitted to the hospital.

(*b*) Patients showing failing compensation in cardiac disease.

(*c*) Anæmic patients. Anæmia was produced by diarrhœa, sprue, ankylostomiasis, ascites, etc.

(*d*) Urological cases with obstruction in the excretory passages, comprising patients with senile hypertrophy of prostate, renal and vesical calculi and pyonephrosis and hydronephrosis.

II. Technique:

Tests were done in the morning before breakfast. At nine o'clock the subject emptied his bladder and that urine was discarded. The urine for examination was collected at 10 and again at 11 in one flask. A sample of blood (0·2 c.c.) for blood urea estimation was taken at 10 o'clock. In each case if the patient did not empty his bladder at the precise time, a careful note of the actual time was made. In this way the correct quantity of urine excreted per minute was measured. The following observations were then made.

U=Urea concentration in urine (mgm. of urea per 100 c.c.)

V=Urine volume (c.c. per minute).

B=Urea in blood (mgm. per 100 c.c.)

C_m =Volume of the blood free from urea per minute, this being for a normal adult of average size about 75 c.c. of blood per minute.

$C_m = \frac{UV}{B}$ (maximum clearance). When the urine volume is less than 2 c.c. per minute, then

$C_s = \frac{U\sqrt{V}}{B}$ =(standard clearance). The results are expressed in terms of c.c. of blood cleared of urea per minute or in terms of percentage of average normal clearance.

Per cent of average normal $C_m = 1{\cdot}33 \frac{UV}{B}$

Per cent of average normal $C_s = 1{\cdot}85 \frac{U\sqrt{V}}{B}$

Urea concentration factor is obtained by dividing the urine urea content by blood urea content. The quotient gives the number of times the kidney concentrates this substance. The figure gives a rough check as to whether the bladder is fully evacuated or not.

Cases with enlarged prostate were always catheterized as errors may arise due to incomplete emptying of the bladder as shown by the dissociation of the values of U.C.F. and C_s.

Blood urea and urine urea were estimated colorimetrically. In cases of nephritis, uric acid was also determined. King and Haslewood technique was adopted.

Macleans urea concentration test was done as far as possible in diseased cases.

Hæmoglobin percentage was estimated by Sahli's method in nephritics and in anæmic cases red blood cell count was also done.

Other biochemical findings such as cholesterol, diastase and protein content of blood of a few cases of nephritis were also noted.

Observations

These observations include 150 cases, 50 normals 100 abnormals.

1. Normals

Taking cases in this group to be the standard normals, the findings are as follows:—

Sub-groups.	Blood urea mg. %		Urine urea mg. %	U. C. F.		Cs.		Function %	
	R.	A.		R.	A.	R.	A.	R.	A.
1.	20-35	26	1660	30-80	64	30-60	50	80-110	94
2.	18-24	21	1600	50-100	80	40-64	57	80-118	100
3.	20-35	31	2100	60-200	79	40-60	31	70-110	97

R=Range. A=Average.

The following are the average figures for healthy Indians between the age period of 15 and 40 according to my observation on 50 cases. They are compared with those of Kasliwal who used Maclean's aeration method of estimating blood urea. His results were based for the normals on 16 medical students and 27 ophthalmic ward patients. The age period in his cases varied from 10-65 years.

Estimations	Extreme Range	Average Range	Average
1. *Blood urea*			
Present observations ..	18-35	20-30	26
Kasliwal's „ ..	12-42	14-30	23
2. *Urea clearance*			
Present observations ..	30-65	45-55	51·6
Kasliwal's „ ..	35-105	40-80	57
3. *Urea concentration factor*			
Present observations ..	36-100	60-80	70
Kasliwal's „ ..	39-120	50-90	65
4. *Percentage functions*			
Present observations ..	70-120	80-100	96
5. *Urine urea*			
Present observations ..	600-2100	1200-1800	1750

The comparison shows that on an average the colorimetric method gave higher blood-urea readings than did the aeration method in the normal cases. The range with the other method, however, was greater possibly because the age variation was wider. On an average normal blood urea figures according to age group vary as given below.

Below 20 years=22 mgm. % or less.
Between 20-30 years=25 mgm. % or less
„ 30-40 years=30 mgm. % or less.
„ 40-50 years
or above=30-35 mgm. % or less.

As the age advances the blood urea content increases and this is in accordance with the changes in the renal vessels that occur on prolonged exposure to the toxic products excreted through this organ in one's life. The increase in the protein intake as the age advances in accordance with the energy requirements accounts also for this increase in the blood urea.

The normal figures for the Europeans and Americans for all the groups are:—

Estimations	Extreme Range	Average Range	Average
Blood urea ..	12-52	20-40	30
Urea clearance ..	45-90	..	55
Urea concentration factor ..	60-80 and upwards		

The results of my observations are shown in Chart I and comparison made with the figures obtained by other workers.

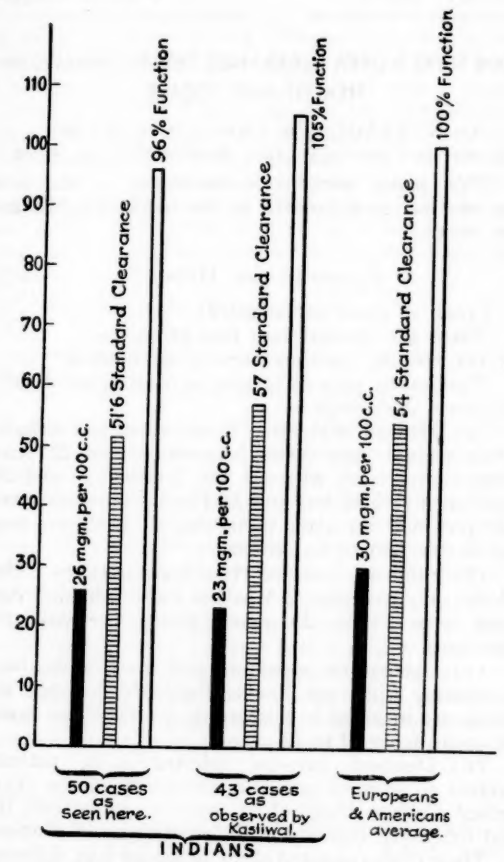

Chart 1—Showing Comparative Representation of Average Blood Urea, Urea Clearance and Percentage Function

There is not the least doubt that in Indians the blood urea is lower on an average than that of Europeans, the average range being 20-35 as compared with 20-40 mg per 100 c.c. This difference probably expresses the type of protein consumed by the two groups of persons. Indeed, here, even those who take animal protein do not take it regularly. If we take the variation of blood urea from 20-35 in normal persons the proteins in the diet on an average ought to be 1·1 gm per kilogram body weight per day. For a person weighing 65 kilograms or 143 lbs. the protein should be about 72 gm. per day. "The ratio N:U=1, where N is equal to the nitrogen metabolized per day in gm. and U is the blood urea in mgm. per 100 c.c." If we calculate the food values of the diet of the medical

students we get not more than 50 gm. per day. Taking 50 gm. as an average of our diet proteins we ought to have a lower range of blood urea than 20-35 mgm. per 100 c.c. and this can be explained on the following hypothesis:

A person residing in a cold climate passes more urine in 24 hours than one in the tropics. The kidneys are regularly flushed by the passage of a large volume of urine in the European countries than is possible in the tropics where there is a more concentrated urine on account of the more evaporation of the moisture as sweat from the exposed skin surface. When the kidneys pass more concentrated urine they have to work under the less favourable circumstances in respect to their excretory function. One can judge this factor of urinary volume from my observations on 50 cases that except in one there was no other case who gave the maximum clearance figure for the calculation of urea clearance.

From my observation on 50 cases in the normal healthy individuals the two figures for the Hindus and Muslims can be separated as follows:—

Estimations	*Muslims (11)*	*Hindus (39)*
Blood urea ..	.. 27·3	25·6
Urea concentration factor	.. 69·6	70·0
Urea clearance ..	.. 50·5	51·9
Percentage function ..	.. 93·0	96·0

This does not mean that the blood urea is generally on the higher side in the Muslims than in the Hindus, and the small difference can be accounted for as an experimental error.

Taking the sub-group separately, one finds that in the medical students the percentage function is lower than in the students of lower classes. It is strange that in laboratory workers the function is better than in medical students, contrary to expectations.

Taking the lower limit in Indians for urea concentration factor and urea clearance as 50 and 40 respectively, if all the observations in the normals and abnormals are recorded, as on Chart 2, one finds that all the observations that fall below this lower normal limit belong to the pathological group. It is also noticed that the extent of the damage done in the diseased cases is in the following order as regards their severity:

1. Nephritics.
2. Urological cases.
3. Heart failure cases.
4. Anæmic patients.

Chart 2 shows these points very obviously. The number of the cases from the pathological group that falls in the normal range is as follows:—

1. Nephritics .. One case.
2. Urological .. Two cases.
3. Heart failure .. Seven cases.
4. Anæmic .. Eight cases.

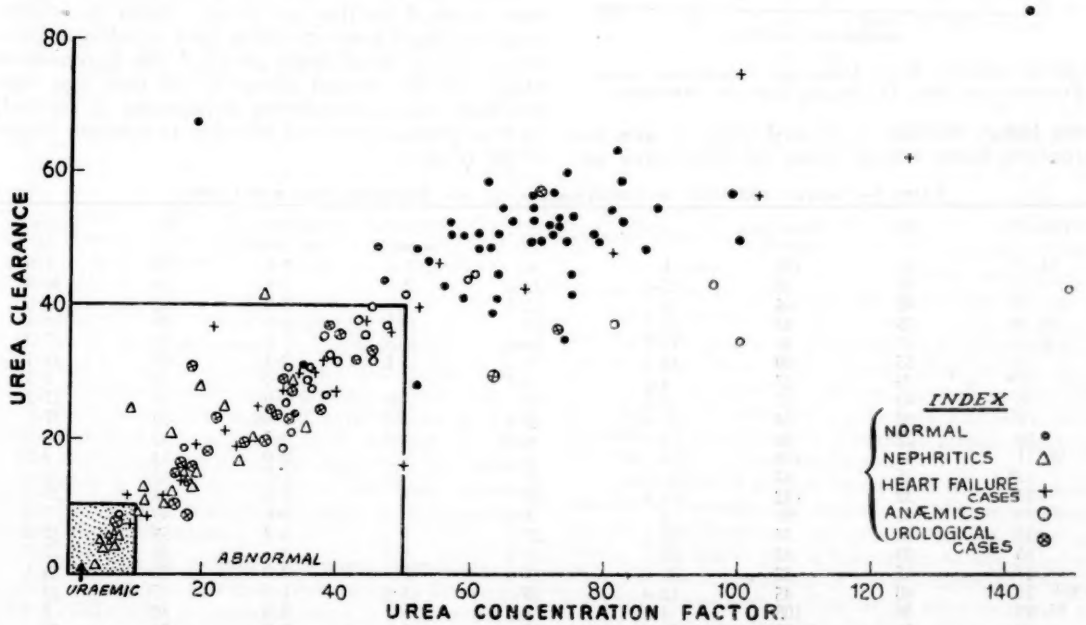

CHART 2—SHOWING UREA CONCENTRATION FACTOR AND UREA CLEARANCE IN HEALTH AND DISEASE

Valley Radot (1938) mentions that normally there is a good deal of flexibility in clearance test and its fixation on the lower side denotes damage of the excretory organs.

II. ABNORMALS

(A) *Nephritics*: (1) Blood urea—Blood urea content and the percentage kidney function have been charted to show their relationship to each other. It is

evident from Chart 3, that mere blood urea estimation is not the real criterion of renal efficiency. In 25% cases of nephritis it remains within the normal limits

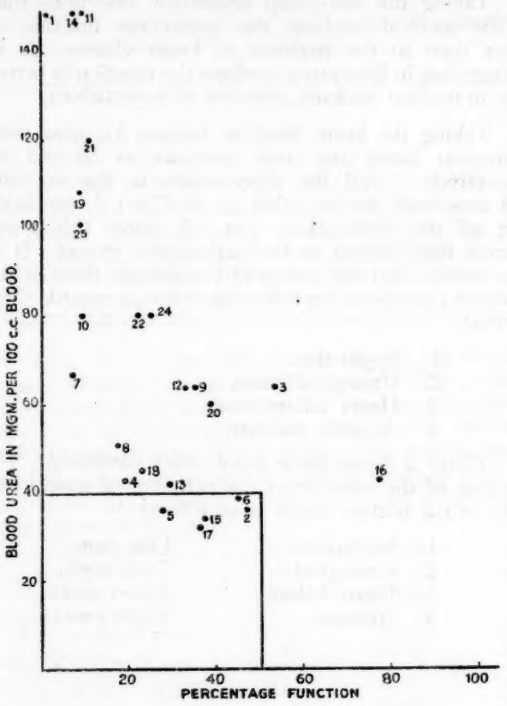

CHART 3—SHOWING BLOOD UREA AND PERCENTAGE KIDNEY FUNCTION BY UREA CLEARANCE TEST IN NEPHRITICS

but the kidney function is lowered while in none was the function found normal when the blood urea was high. This chart shows that in two cases the blood urea was found high even when the function was between 50 and 60%. Thus below 80%, the function is considered slightly impaired while below 50% the defect is quite enough to raise the blood urea in a good majority of cases. It is also evident that the blood urea level in Indians is lower than in Europeans, as in about 20% cases it was found between 30 and 40 mgm. per 100 c.c. even in cases of nephritis (normal for the standard European figures).

Hæmoglobin content in the six cases which showed normal blood urea was as follows:—

Case No.	*Blood urea*	*Age*	*Hb. %*	*Remarks*
2	36	25	69	
5	36	25	91	
6	39	35	80	
15	34	30	35	Malaria with ankylostomiasis.
17	32	45	20	Ankylostomiasis.
23	40	40	46	Ankylostomiasis.

Blood urea in the last three cases was lower no doubt but the hæmoglobin content of these cases was affected too much by the accompanying hookworm infection and the increase in the plasma content in such cases might account for both. In the first three cases the hæmoglobin content was between 70 and 90% and the blood urea was not very much raised. These three cases were young adults between 25 and 30 years of age. Blood urea in normal healthy persons of this age should not be more than 30 mgm. per 100 c.c. and so these figures were on the high side considering our own standard for this age group. Hence the conclusion that blood urea estimation itself is sufficient proof of the kidney insufficiency provided this is considered along with the clinical picture of the case, age, race, residence, and accompanying disturbances in the body such as anæmia, protozoal infection (a common feature of the tropics).

TABLE I—SHOWING RESULTS OF INVESTIGATION ON THE NEPHRITIC GROUP OF CASES

Case No.	*Age*	*Blood urea mgm.%*	*Cm or Cs.*	*U.C.F.*	*Uric acid mgm.%*	*Maclean urea conc. test %*	*Hb %*	*Function %*
M. 1*	60	195	1	4	9	0·9	91	1·8
2	25	36	25·3	22·2	2	2·5	69	46·8
3	40	64	29	32·8	3·6	3·0	62	53·6
F. 4*	35	43	10·5	14	2·8	1·0	50	19·4
F. 5	25	36	14·7	16·6	2·5	1·8	91	27·2
6	35	39	24·4	9	2·4	2·5	80	45·1
7*	30	67	4·1	6·7	5	0·9	51	7·5
8	40	51	9·3	10	3·8	1·0	53	17·2
9	30	64	21·95	35·4	3·5	1·65	60	37·1
10	22	80	5	6·25	4·5	0·8	52	9·25
M. 11	35	178	5	6	6	0·9	53	9·25
12*	25	64	17·75	25	3·5	1·7	47	32·6
13	32	42	15·8	19	3	1·6	38	29·3
F. 14	45	200	4	5·2	7	0·8	49	7·4
15	30	34	20	27·1	3	3·2	55	37·0
16	20	43	42	29·3	3·2	3	88	77·7
17	45	32	28	19·6	3·5	0·9	20	36·1
MF. 18	40	45	12·6	18	3·6	1·5	63	23
M. 19	36	108	4·4	5·5	6	0·8	50	8·1
20	36	60	21	15	4	1·9	75	39
F. 21	28	120	5·9	6·6	5	1	55	11
M. 22	50	80	12	15	4·5	1·2	70	22
23*	40	75	10·6	10·6	..	1·2	45	19·6
M. 24	35	80	13·4	11·2	4	1·3	65	24·8
25*	36	101	5	6	5	0·8	45	9

M=Muslim. F=Female. * Died in hospital.

(2) Hæmoglobin content of blood and function percentage in nephritis—Plotting these two figures, as on Chart 4, one sees that there is a definite

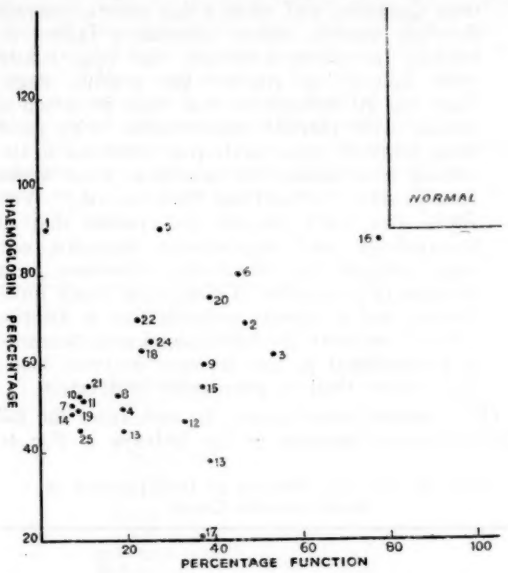

CHART 4—SHOWING HAEMOGLOBIN RELATIONSHIP WITH EXCRETORY FUNCTION IN NEPHRITICS

relation between Hb% and the functional capacity of kidneys in nephritis. Considering the function the cases fall as given below, between 60-80% one case only, 40-60% three cases, 20-40%, nine cases and below 20%, twelve cases. Most of the cases show hæmoglobin between 50 and 70%. There are few exceptions as well, as case no. 1 and 5 which show marked lowered renal function with hæmoglobin content altogether unaffected. In these cases the duration of illness was too short to affect the hæmoglobin content of blood. When there is accompanying hookworm infection the hæmoglobin is affected remarkably as in cases no. 15, 17 and 25.

Ashe of New York suggested that Hb% and R.B.C's are both decreased in cases of renal insufficiency but in cardiac and cardio-renal cases the R.B.C. count is normal while hæmoglobin content may be diminished, whereas in cases of hypertension both the Hb% and R.B.C. count are either normal or above it. In diffuse glomerulotubular nephritis, the anæmia is probably a result of injury to the bone marrow, which shares the constitutional damage that is suffered by the heart and retina.

Thus there is definite prognostic significance of anæmia in cases of nephritis. According to Brown and Roth (1923) with no anæmia there is 18% mortality in 2½ years. With 60-80% Hb, there is 46% mortality, and with less than 60% Hb 85% mortality in 2½ years.

(3) Uric acid content of blood in nephritis—Figures for uric acid and blood urea have been charted for comparison, as seen in Chart 5. Here it is seen that blood urea goes high in nearly all the cases, while uric acid in 8 cases only. No staircase phenomenon was observed as sugested by Myer (1929) that first uric acid then blood urea rises.

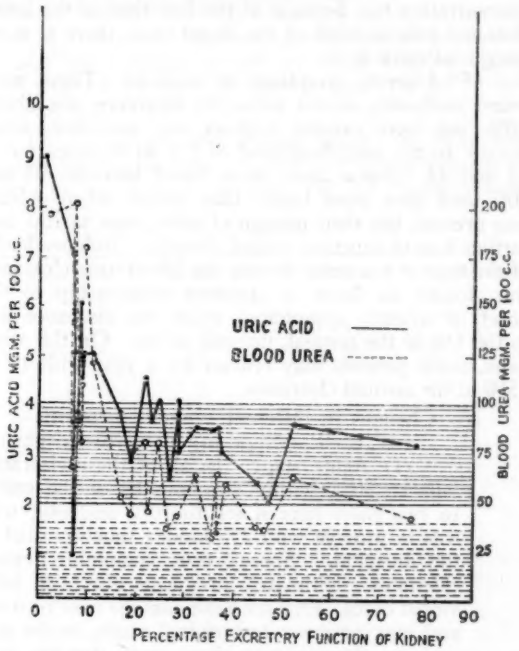

CHART 5—SHOWING URIC ACID AND BLOOD UREA IN NEPHRITICS

(4) Urea concentration test—Taking 2·5% as 100% with Maclean's urea concentation test, we get below 40% function in 44% cases of nephritis, with this method, while urea clearance shows that in about 80% cases the function is reduced to less than 40%. This shows that urea clearance test is a better guide of kidney function than urea concentration test alone.

Personally, I saw that the results with Maclean's concentration varied much, for much depends on how the patient carries out the directions given to him. In the terminal stages of nephritis with high blood urea content, a urinary urea concentration within normal limit may be observed despite reduced renal ability, as case no. 15 with 3·2% Maclean and 37% kidney function. Hence this is uncertain unless blood urea and the urine volume are known.

In cases of nephritis, blood urea has got a definite relation to the urea clearance test in inverse proportion. As the urea clearance test goes down the blood urea rises. This might give a temptation to use blood urea alone for the information of kidney damage, instead of the complicated test of Van Slyke urea clearance test. There is not the slightest doubt that the former is a much more sensitive indicator of renal efficiency than the latter, as found by Van Slyke (1921). High blood urea can be found in other diseases such as pneumonia, intestinal obstruction, after ingestion of large quantity of urea, and even after a high protein diet. These should be kept in mind when considering its value.

MacIntosh, Moller, and Van Slyke (1928) are of opinion that the urea clearance test is even better than blood creatinine estimation and phenolsulphonephthalein test and also more accurate than urea concentration test because of the fact that as the latter does not take account of the blood urea, there is more margin of error in it.

(5) Uræmic symptoms in nephritis—These were found uniformly absent when the clearance was above 10% but were present without any exception when it sank to the neighbourhood of 5% as in cases no. 1, 11 and 14. Some cases were found between 10 and 20% and they were better than before when œdema was present, but their margin of safety was narrow and further loss of function caused uræmia. Independently of the type of the renal disease the blood urea clearance was found to have a constant relationship to the onset of uræmic symptoms, when the clearance fell to the 5% of the normal, uræmia set in. On the other hand, some patients may remain for a year with 10 to 15% of the normal clearance.

(6) Changes in different types of nephritis—

(*a*) Hæmorrhagic and glomerular nephritis.

(*i*) Acute stage. In all of these clearance falls to 50% within two months of the onset. In two cases here it fell to 10% and one, who was in uræmia with 2% kidney function died in the hospital. Some cases maintain normal clearance. Ultimately prognosis differs in individual cases. One showing damage may recover, and may progress to terminal stage, or the one with no damage may show great damage in a very short time. Good prognosis is seen in those cases which recovered within 4 months, and not in others. Cases no. 1 and 14 belong to this group. Out of these the first died in the hospital and the other in whom the prognosis was not good was taken away against medical advice by the relations.

(*ii*) In the chronic active stage it was always found less than 60% and usually less than 40%. It may remain stationary in few cases but in others it always follows the downward course. Most of the cases investigated here belong to this group. Excluding a few cases falling in other groups, all can be put under this head.

(*iii*) In the terminal stages, shown by accumulation of urea and loss of any tendency to variation in clearance, it was found below 20%. Out of a good many cases in whom the clearance fell permanently to below 20%, many died in the hospital. Cases no. 7, 11, 19, 21 and 22 belong to this group.

(*b*) Nephrosis. Blood urea clearance falls eventually in these cases below normal, except in a few cases where the condition is temporary. In all the cases where it is found below normal it progressively decreases till it comes to the level where uræmic symptoms might be seen as in the other two groups. Only one case amongst these was noticed and the percentage function in him was 37.

(*c*) Arteriosclerotic Bright's disease. This may continue for four years (benign) (Volhard and Fahr, 1918) without lowering the blood urea clearance, and when a fall occurs, uræmia develops quickly, unless circulatory failure or vascular accidents terminate the case before renal failure has reached the uræmic stage. Case no. 10 belongs to that class in which a patient with marked hypertension, with good renal function, shows a drop of clearance to the uræmic level within few months or even weeks (malignant) (Volhard and Fahr *loc. cit.*). Van Slyke (*loc. cit.*) formed the opinion that in hæmorrhagic and degenerative nephritis one may interpret the blood urea clearance as a measure of proportion of glomerular tissue functioning, and it appears probable that in arteriosclerotic nephritis the fall in blood urea clearance is proportional to the decrease in renal blood flow rather than to glomerular obstruction.

(B) *Cardiac failure cases*—In such cases the fall in the excretory function of the kidneys is due to lessened circulation in them. In advanced cases of essential hypertension urea clearance gives low reading for renal function, due to arteriosclerotic changes in the renal vessels, and again due to diminished blood flow in them. Only when the decompensation begins in cases of heart failure, is the decrease in renal function seen (Table II).

TABLE II—SHOWING RESULTS OF INVESTIGATION IN HEART FAILURE CASES

	Case No.	*Blood urea mgm.%*	*Cm or Cs.*	*U.C.F.*	*Maclean's urea concentration test %*	*Function %*
	1	33	30	34	2·4	63
	2	75	18·5	19·8	2·4	36·6
	3	32	37·5	21·75	2·6	40
	4	43	11·5	9·5	3·5	17·5
M.	5	99 (180/120)	11·1	8·6	1·3	16
	6	53·5 (170/108)	50	16·1	1·2	30
	7	49 (220/180)	14	10·8	2·0	20
M.	8	54	16·6	14·1	1·8	26·1
M.	9	27	55·5	46·6	2·2	86·2
	10	33	39·4	27·6	2·1	43·1
	11	36	23·6	20·7	2·1	38·4
M.	12	28	125	63	2·4	116
	13	36	80·5	48·3	2·25	81
	14	36	102·8	56·5	2·5	104·6
F.	15	31	67·7	43	2·1	80
	16	21	100	75	2·0	138
M.	17	107 (185/120)	9·3	7·5	2·1	14
	18	62	25	19·5	1·6	36
	19	38	28	25	1·65	46·25
	20	42	32	28	2·0	51·8
	21	36	52	40	2·1	74
F.	22	31	48	36	2·1	66·6
M.	23	45	38	32	1·7	59·2
	24	37	44	38	2·25	70·3
	25	38	36	30	..	55·5

M=Muslim. F=Female.

(C) *Anæmic cases*—When the hæmoglobin begins to fall from 80% downward the kidney function also

shows lowering from 80 to 40% along with the fall in blood urea, showing that it is not the nitrogen retention which causes decrease in function, but the difference in the quality of the blood supplied to the kidneys. Here, as seen in Table 3, there were 19 cases who showed hæmoglobin between 50 and 90%, and out of these the function was between 40 and 80% in 16 cases. This roughly gives an idea that every 10% fall in hæmoglobin content of blood gives 10% fall of the renal efficiency, in cases of anæmia (Table III).

(D) *Urological cases*—(*i*) In cases of enlarged prostate the figures for blood urea should be supplemented with the urea clearance figure in judging it to be fit for a single stage operation or a two-stage one. 60% is the limit, under which two stage operation should be done (Table IV).

(*ii*) Blood urea is not so much raised in ordinary cases of vesical calculus as it is in renal calculus cases, and the clearance test informs us of the extent of the damaged kidneys (Table V).

(*iii*) In acute inflammatory cases such as pyonephrosis the function is not so much lowered as in pynonephritis cases, as is seen in Case no. 17 in this group (Table VI).

Urea clearance test should always be taken into consideration when the question for descending pyelography arises and in this way, the life of the patient

TABLE III—SHOWING RESULTS OF INVESTIGATION ON PATIENTS WITH ANAEMIA

Case No.	*Diagnosis*	*Blood urea mgm.%*	*U.C.F.*	*Cm or Cs.*	*R.B.C.% 5 mil.=100%*	*Hb*	*Function %*
1.	Anæmia with ascites	28	32	19	60	53	35
2.	Diarrhœa	21·6	32·6	31·2	32	30	58
3.	Anæmia with diabetes	42·8	7	9·8	32	29	18·1
4.	Diarrhœa	38	47·4	37·4	75	72	69
5.	Anæmia with diabetes	36	33·3	21·0	60	59	38·8
6.	Anæmia	36	80·5	37·8	80	75	69·7
7.	Sprue	23	100	35	72	70	64·75
8.	Anæmia	25	96	43·8	64	66	81
9.	Anæmia with ascites	24	16·6	19·3	63	55	35
10.	Ankylostomiasis	32	39·4	27·2	25·2	28	50·3
11.	Anæmia	21	162	43	72	70	80
12.	Sprue	38	43·4	38·2	64	50	71
13.	Anæmia	35	45	32	72	71	59·2
14.	do.	32	36	30	70	68	55·5
15.	do.	28	38	36	80	78	66·6
16.	do.	33	40	32	65	61	59·2
17.	do.	25	45	40	75	73	74
18.	do.	28	36	28	65	60	51·8
19.	do.	29	32	26	60	56	48·1
20.	do.	30	60	45	82	81	83·3
21.	do.	36	50	42	70	69	77·7
22.	do.	28	38	32	66	65	59·2
23.	do.	32	44	36	68	65	66·6
24.	do.	38	60	45	85	84	83·25
25.	do.	30	36	30	64	63	55·5

may often be saved by judging the true state of the other kidney.

CONCLUSIONS

Following are the observations based on the colorimetric method of estimation of urea in blood and urine:

(1) Blood urea as observed here amongst the 50 absolute normal cases vary from 18-35 mgm. per 100 c.c., a figure lower than that for Europeans. The average figure is 26 mgm. per 100 c.c. Blood urea increases as the age advances, and its amount varies with the protein consumption of different people. The figures for Muslims and Hindus do not vary much and the slight difference as is seen here can be accounted for as being within the range of the experimental error.

(2) Urea clearance for the people of this part of our country varies from 30-65. The average figure is 51·6. Maximum clearance is not commonly seen here. Only one case out of 50 was observed. In that case the figure was 68 in place of normal 75.

(3) Urea concentration factor varied from 36 to 100. The average figure is 70.

TABLE IV—SHOWING RESULTS IN CASES OF ENLARGED PROSTATE

Case no.	*Blood urea mgm. %*	*U.C.F.*	*Cm or Cs.*	*Maclean's U.U.C. Test %*	*Function %*
5.	90	20·4	18·5	1·5	34
7.	68	16·6	16·3	1·7	30
10.	85	72·7	36·3	3·0	67·5
14.	56	26	20	1·7	37
18.	38	32	29	2·5	54
20.	100	7	8	0·9	14·8
23.	80	18	16	1·4	29·6
24.	44	40	36	2·2	66

Blood urea is increased in nearly all the cases.

TABLE V—SHOWING RESULTS IN CASES WITH RENAL AND VESICAL CALCULI

Case No.	Diagnosis	Blood urea mgm. %	U.C.F.	C_m or C_s	Maclean's U.U.C. Test %	Function %
1	Vesical calculus	27	37·6	24·8	..	46
2	Renal calculus	51	16·1	15·6	1·5	29
3	,,	65	18·2	30·8	1·6	57
6	,,	60	30	25	..	46·3
9	,,	28	39	37	..	68
11	,,	52	22	24	1·6	44·4
12	,,	53	30	25	1·6	46·25
16	Vesical calculus	54	17	9·5	..	17·5
19	Renal calculus	48	32	28	..	52
21	,,	58	16·5	14	..	26
22	Vesical calculus	34	29	20	..	37

TABLE VI—SHOWING RESULTS IN CASES WITH INFLAMMATORY AND DEGENERATIVE CONDITIONS OF KIDNEYS

Case No.	Diagnosis	Blood urea mgm. %	U.C.F.	C_m or C_s	Maclean's U.U.C. Test %	Function %
4	Pyonephrosis	37	42·7	32·6	1·2	60
8	do.	51	70	57·2	..	106
13	Cystic kidney	46	63	35	..	64·75
15	Renal tumour	62	32	24	2·2	44·4
17	Pyonephritis	45	15	11·4	..	21
25	Hydronephrosis	39	45	35	1·7	65

(4) Kidney function varies in normal cases from 70 to 100, the maximal range being 70-120%. The average figure is 96%.

(5) 50 is the lower limit for urea concentration factor and 40 for urea clearance in Indians. Figures below these definitely indicate renal damage.

(6) The extent of damage in four different groups as regards severity was found in the following order:—

(*i*) Nephritics—maximum. (*ii*) Urological cases and (*iii*) cardiac failure cases—medium. (*iv*) anæmics —minimum.

(7) It was observed that as the blood urea rises the urea clearance figure and the functioning capacity of the kidneys definitely decrease, but, in a certain proportion of cases the blood urea may remain normal in spite of the lowered renal function. Figures for blood urea should be considered in relation to the clinical picture of the case, his age, race and other accompanying variations in the body.

(8) In cases of nephritis as blood urea rises hæmoglobin content of the blood also lowers down, and this is of prognostic significance here. Figures lower than 40% are not of good omen.

(9) Urea clearance test is of great value in cases of nephritis. When the percentage function is 10, uræmia is uniformly present, and the end is in view.

(10) Urea clearance test is of special value in judging the progress of the case from day to day when under treatment in a big hospital where such facilities are available. The technique, though at the outset it seems to be an elaborate one, is not much more difficult than the urine urea concentration test. This is specially so when the simplified technique of Van Slyke and Cope (1931) is adopted, which gives us a direct reading as regards the percentage of the kidney function without much mathematical calculations.

(11) Uric acid and other biochemical examinations are not of much use in routine practice. They are only of prognostic significance. Uric acid was found raised only in a limited number of cases, and not in all those where urea was high.

(12) (*a*) In acute stage of glomerular nephritis the clearance falls a great deal, even to 10%, within two months of the onset. Ultimately prognosis differs in individual cases. (*b*) In chronic cases the clearance is always found lower than 60% and usually even less than 40%. (*c*) In the terminal stages with accumulation of urea, it is lower than 20% and, when 10% or lower, uræmia is always seen.

(13) In arteriosclerotic Bright's disease the clearance falls suddenly to the lower limits where uræmic symptoms are commonly seen.

(14) In cases of nephrosis the clearance in the beginning is not much lowered but in the long run it also gradually falls down to the range of uræmia *i.e.*, 10%, and this result is generally seen after quite a long interval.

(15) Urea clearance is lowered when decompensation begins in cardiac cases and the extent of damage is not great. The function is more likely to recover when compensation takes place.

(16) In cases of anæmia due to the changes in the quality of the blood, the clearance is diminished. On an average with every 10% fall in hæmoglobin content a 10% fall in urea clearance course.

(17) In cases of enlarged prostate, urea clearance is of great help in judging whether they are fit for one-stage operation. When the urea clearance figure is below 60% the indication is for a two-stage operation.

There is more damage in renal calculus cases than in vesical ones, except when the stone in the bladder is large enough to cause back-pressure.

(18) The value of renal efficiency tests should be judged from all aspects as a certain figure normal for one state may be quite abnormal for another.

REFERENCES

ADDIS, T.—*J.A.M.A.*, **84**:167, 1925.
Idem—Ibid., **85**:1013, 1925.
ADDIS, T. AND GOLDING, W—Medical Annual, 1933, p. 394.
BEAUMONT, G. E. AND DODDS, E. C.—Recent Advances in Medicine, 1936, p. 386.
BROWN, G. E. AND ROTH, G. M.—*J.A.M.A.*, 1923.
EPSTEIN, ALBERT—*J. A. M. A.*, **69**:444-467, 1917.
HARRISON, G. A.—*Brit. J. Exper. Path.*, **3**:28, 1922.
KASLIWAL, R. M.—Thesis for the M.D. Lucknow University.
KING, E. J. AND HALSLEWOOD, G. A. D.—*Lancet*, **1**:886, 1937.
MACLEAN, H. AND DE WESLOW, O. L. V.—*Brit. J. Exper. Path.*, **1**:53, 1920.
MCINTOSH, J. F., MOLLER, E. AND VAN SLYKE, D. D.—*J. Clin. Invest.*, **6**:467, 1928.
MYER—Medical Annual, 1929, p. 408.
PETERS, J. P. AND VAN SLYKE, D. D.—Quantitative Clinical Chemistry, 1931, Vol. 1, p. 353.
VALLERY RADOT—*J.A.M.A.*, **110**:2183, 1938.
VAN SLYKE, D. D.—*J. Biol. Chem.*, **46**: 91, 1921.
VAN SLYKE, D. D. AND COPE, C. L.—Quantitative Clinical Chemistry, 1931, Vol. 2, p. 935.
VOLHARD F. AND FAHR, T.—Barlin, 1918, p. 168.

SOME OBSERVATIONS ON CASES OF ASCITES IN HYDERABAD-DECCAN*

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(Continued from previous issue)

II. ASCITES ASSOCIATED WITH CIRRHOSIS OF THE LIVER

An analysis of 343 cases of ascites tabulated in Part I shows that ascites was associated with cirrhosis of the liver in 127 or 37% of cases. Out of these 127 cases 108 or 85% were classed as portal cirrhosis, 13 biliary cirrhosis (adults and children) and 6 syphilitic hepatitis (Table I). In this part of the paper observations on cases of portal cirrhosis will be presented and discussed.

TABLE I—SHOWING CLASSIFICATION OF 127 CASES OF ASCITES ASSOCIATED WITH CIRRHOSIS OF THE LIVER.

S. No.	Diseases	No. of cases	Recovered	Improved	Discharged otherwise	Died
1.	Portal cirrhosis ..	108	2§	7	67	32†
2.	Biliary cirrhosis ..	7	..	1	2	4‡
3.	Infantile biliary cirrhosis	6	..	..	3	3
4.	Syphilitic cirrhosis ..	6	1	4	1	..
	Total	127	3	12	73	39

§ Ascites in both of these cases had completely disappeared without necessitating abdominal paracentesis; one of these was re-admitted after one year with signs of advanced liver failure and died after a few paracenteses; the other was re-admitted after about a month and died within a week; a postmortem examination on the latter revealed cirrhosis of the liver.

† A postmortem examination was performed in 8 out of 32 fatal cases and all of them showed portal cirrhosis.

‡ A postmortem examination was done in one case of biliary cirrhosis and diagnosis confirmed; this case also showed a fair number of abscesses in the liver near the portal tracts—? terminal infection.

Age and Sex—While a few cases were seen among children and adolescents, the condition was preponderatingly common in adults and the middle-aged; 70% of the cases occurred between 30 and 50 years of age while the proportion of persons of this age group to the total population of the State was only 23%. This is in agreement with earlier studies in India (Rao, 1933; Menon, 1935). Males were much more frequently affected than females, the ratio being 2·8:1 whereas their relative numbers in hospital in-patients were nearly equal. This observation again is in conformity with that of others (Rolleston and McNee 1929; Rao, 1933; Menon, 1935; Ratnoff and Patek, 1942). Wayburn and Guerard (1940) have, however, noted a preponderance of women in cases of cirrhosis of the liver associated with neuropathy.

Caste—The majority of the sufferers were Hindus (Hindus 80·5%, Muslims 15·7%); the relative numbers of Hindus and Muslims in hospital admissions were nearly equal. If the incidence were compared on these figures it would appear as if the disease was five times as common among Hindus as among the Muslims, but in proportion to their relative numbers in the State population (84·3% and 11·3% respectively) there seems to be no selective affection.

Occupation—Most of the patients were poor class cultivators, farm-labourers or village artisans. This is only to be expected since a large majority of the patients were from rural areas.

Geographical Distribution—As stated in Part I, on classifying the patients according to the districts to which they belong, it was seen that the number of patients coming from a given area depended on nearness of the place from Hyderabad city and ease of access thereto. The figures, therefore, fail to show the real incidence of the condition in different parts of the State.

Dietetic History—The dietetic history of most of the patients was the same as described for cases of Nutritional Ascites, the diets were commonly lacking in milk or milk products and other protective foods (Table H, Part I).

Out of 108 patients 20 were total abstainers by virtue of their religion (Brahmin, Lingayat, etc.); 40 gave a history of occasional 'toddy' or 'sendhi' drinking (the alcohol content of these, however, is only about 7%), while no definite history of alcohol addiction was obtained in the rest (Table II).

TABLE II—SHOWING DIETETIC HISTORY OF 108 CASES OF PORTAL CIRRHOSIS.

Vegetarian and abstainer		20 cases
Non-vegetarian		
(*a*) History of drinking 20-30 ozs. of 'toddy' or 'sendhi' once or twice a week in	40	
(*b*) No history of alcohol habit in	48	
		88 cases

Antecedent Conditions—A history of of antecedent attacks of fever of short duration was obtained in 46 cases, and of diarrhœa in 13, while in the rest the onset was characterised by a gradual enlargement of the abdomen and increasing weakness without any obvious cause. Three patients gave a history of ascites with complete recovery 1, 5 and 23 years before admission. A definite history of previous attack of jaundice was obtained only in three cases.

The duration of ascites before the patients sought admission into the hospital varied from less than a month to four years, but in the majority of the cases it was between one to six months.

Clinical Features—The patients sought admission into the hospital mainly for the relief of enlargement of the abdomen, weakness and scanty urine. In the majority of the cases they were extremely weak and emaciated; the skin was dry and inelastic and the muscles were generally wasted. The body weight after the removal of fluid by paracentesis was low; in most of the cases it was 80 to 100 lbs. Inspite of a large accumulation of fluid in the peritoneal cavity marked dyspnoea was noted only in 21 cases. Scantiness of urine was complained of by 81 patients.

* Part 2 of a paper presented before a meeting of Medical and Veterinary Section of the 32nd Annual Session of the Indian Science Congress held at Nagpur in the first week of January 1945.

The findings on clinical examination are shown in Table III from which it will be seen that ascites was associated with oedema of the lower limbs in 92 cases and pleural effusion in 9; pericardial effusion was not noted in any of the cases. Jaundice was observed only in 15 cases and occasional bouts of low fever in 18. The veins of the anterior abdominal wall and lateral aspect of the chest were prominent in 55 cases, but typical caput Medusae was not observed in any case. The hæmorrhagic phenomena were rare; hæmatemesis in one case, melena in three, epistaxis in one and bleeding piles in five cases. Only two patients had vascular spiders and one had clubbed fingers. Gynaecomastia was observed in one case. There was no hernia in any of the cases.

TABLE III—SHOWING CLINICAL SIGNS IN 108 CASES OF PORTAL CIRRHOSIS

S. No.	Signs	Number of cases
1.	Ascites	108
2.	Oedema	92
3.	Pleural effusion	9
4.	Pericardial effusion	nil.
5.	Fever	18
6.	Jaundice	15
7.	Prominent abdominal veins	55
8.	Epistaxis	1
9.	Hæmatemesis	1
10.	Melena	3
11.	Hæmorrhoids	5
12.	Varicose veins	nil.
13.	Vascular spiders	2
14.	Hernia	nil.
15.	Clubbed fingers	1
16.	Palpable liver	33
17.	Palpable spleen	55
18.	Tender calf muscles	2
19.	Hypo-reflexia	6
20.	Absent deep reflexes	6
21.	Mental sluggishness	21
22.	Delirium	14
23.	Gynaecomastia	1
24.	Xerosis	1

The heart on clinical examination was found to be displaced upwards but there was no enlargement or dilatation. The pulse in most of the cases was small but regular. The blood pressure in the majority of the cases was low; systolic 80-110 mm. Hg. and diastolic 60-80 mm. Hg; in one case the systolic pressure was only 78 mm. Hg. The average blood pressure of 80 cases was 105/73 mm. Hg. While these figures appear low when compared to western standards the average blood pressure of 12 Bengali young men between 20 and 27 years of age, was 108/78 mm. Hg. (Mukerjee and Gupta, 1938). In Hyderabad, Rahaman (1936) has found the average blood pressure of 32 young men between 19 and 32 years of age to be only $100 \cdot 4 \pm 11/71 \cdot 8 \pm 5$ mm. Hg. Only two cases had blood pressure higher than 160 mm. systolic; one was associated with chronic nephritis; in the other the cause was unknown.

The lungs were clear except for the presence of a few coarse rales in some cases. As mentioned above, pleural effusion was detected in 9 cases.

The liver was palpable in 33 cases. It was generally hard and in some cases rough with uneven surface. The spleen was palpable in 55 cases. Mentally, the patients were rather dull and slow in answering questions; terminal delirium was observed in 14 cases. Signs of peripheral neuritis such as calf-tenderness and hyporeflexia were found only in 6 cases. A large majority of the patients had oliguria, the urinary output in 24 hours being less than 10 ounces.

LABORATORY FINDINGS

Urine—The urine was high coloured with a specific gravity ranging between 1010 and 1020. A trace of albumin without casts was found in 39 cases; moderate quantity of albumin with hyaline casts in 2, and with red blood cells and granular casts in 4 cases, while no albumin or casts were detected in the remaining 63 cases. Bile salts and pigments were found in 7 cases associated with jaundice.

Faeces—Out of 37 cases in which a microscopic examination of the stools was made, ova of ankylostoma were found in 4 cases and ova of ascaris in one case. None of the specimens showed Entamoeba histolytica or evidence of chronic dysentery.

Blood Picture—Anæmia of varying grades was noted in most of the cases (R.B.C's. between 2 to 4 million per c.mm., and hæmoglobin 40 to 70%; Table IV). The anæmia was generally of normocytic and slightly hypochromic type. A slight leucopenia was noted in most of the cases; there was no leucocytosis in uncomplicated ones.

TABLE IV—SHOWING BLOOD PICTURE IN CASES OF PORTAL CIRRHOSIS

R.B.C. per c.mm.	Number of cases	*Haemoglobin percentage.	Number of cases
2—3 Million	8	40—50	6
3—4 „	14	50—60	6
4—5 „	20	60—70	5
Over 5 „	1	70—80	11
		80—90	9
		90—100	6

* Hæmoglobin 100%=13·7 G. per c.c. of blood.

Blood Chemistry—The blood urea was estimated in 13 cases. It was

(1) within normal limits (15 to 40 mgm. per 100 c.c.) in 5 cases,

(2) slightly raised (40 to 50 mgm. per 100 c.c.) in 3 cases, and

(3) high (more than 50 mgm. per 100 c.c.) in 5 cases.

A high blood urea was noted mostly in terminal stages or in cases associated with chronic nephritis. The blood chlorides were estimated in 6 cases; they were within normal limits (365 to 468 mgm. per 100 c.c.) in 5 cases and high in one case (730 mgm. per 100 c.c.). The blood cholesterol was estimated in 5 cases and was found to be within normal limits.

The Van den Bergh test was performed on the sera of 14 patients; of these, clinical jaundice was present in 2, while the rest were non-icteric. The serum of one of the icteric patients gave an immediate direct positive reaction and the bilirubin content was 6 mgm. per 100 c.c.; this patient had fever and leucocytosis and died within a few days after admission. The serum of the other icteric patient gave a delayed direct positive reaction and the bilirubin content was 2·6 mgm. per 100 c.c.; this again was an advanced case of cirrhosis of the liver and was fatal within 7 weeks after admission. Of the 12 non-icteric cases, the Van den Bergh test was delayed direct positive in 4 with a raised serum bilirubin (1·4 to 2 mgm. per 100 c.c.), while in the remaining 8 the bilirubin content was less than 0·5 mgm. per 100 c.c. (normal 0·1 to 0·5 mgm. per 100 c.c.).

The proteins of the serum were estimated in 8 cases (Table V); the averages are, total proteins 5·59 per cent, albumin 2·13 per cent and globulin 3·46 per cent. In all the 8 cases the albumin was less than 3 per cent, the albumin globulin ratio was altered in all and completely reversed in 5 cases.

TABLE V—SHOWING SERUM PROTEINS IN CASES OF CIRRHOSIS OF THE LIVER WITH ASCITES

Serial No.	*Case*	*Total Protein*	*Albumin*	*Globulin*
1.	E.R.	6·37	2·03	4·34
2.	Nar.	3·50	1·90	1·60
3.	G.M.	3·25	1·94	1·31
4.	Cha	7·18	1·87	5·31
5.	Lin.	5·56	2·00	3·56
6.	M. Ib.	5·43	2·37	3·06
7.	Ash	6·56	2·81	3·75
8.	Shi.	6·87	2·12	4·75
Average		5·59	2·13	3·46

Liver Function Tests—The Rose-Bengal dye excretion test was performed on 15 cases. The results showed hepatic insufficiency in all of them, slight in 5, moderate in 2, and high in 8 cases.

Renal Function Tests—Out of 13 cases tested, a high blood urea was noted in 5 cases who had either chronic nephritis or were in terminal stages of liver cirrhosis, while in others it was within normal limits. The phenolsulphonephthalein test showed a low excretion of the dye (2 hours excretion less than 50 per cent) in 7 out of 10 cases. These patients also had oliguria as an important clinical finding. On the other hand, the administration of a mercurial diuretic (neptal 0·5 c.c. intramuscularly) to some of these produced a very good response. In 9 fatal cases which were examined post mortem, the kidneys were found to be normal. Further investigations are necessary to elucidate the cause of oliguria in cases of portal cirrhosis.

The Wassermann Reaction—Out of 74 cases tested, a positive reaction was obtained only in 16 and a positive Kahn's test in one case whose serum was anti-complimentary, *i.e.*, in all 17 or 23 per cent of the cases gave serological evidence of syphilis. It is noteworthy here that in Radhakrishna Rao's (1933) series of 55 cases, a positive reaction was obtained in 41 cases or 80 per cent, and in his opinion syphilis plays an important role in the aetiology of portal cirrhosis. On the other hand, Ratnoff and Patek (1942) in more than 300 cases, found positive serological evidence of syphilis in 16 per cent and conclude that 'syphilis is rather a concomitant factor in cirrhosis, the significance of which is not clear.' It would, however, appear that no inference can possibly be drawn from positive Wassermann reactions obtained in a certain number of cases in the absence of accurate data regarding the prevalance of syphilis in the general population and the social class of a given area from which the patients are drawn.

Character of Ascitic Fluid—While doing paracentesis, the pressure of the intra-abdominal fluid was noted in 32 cases; it was nearly zero in 10, 1 to 2 inches above the auricular level in 7 and 3 to 5" in 14 cases; in one case it was as high as 8". The pressure, however, had no relation to the quantity of fluid and sometimes even a small collection gave a high pressure. Here again, further investigations are necessary to explain the causation and significance of raised intra-abdominal pressure.

The ascitic fluid was a transudate in all the 45 cases examined; the specific gravity in the majority of the cases was between 1010 and 1015, the average protein content 0·43 per cent and chlorides 0·571 per cent. A positive, delayed direct or indirect, Van den Bergh reaction was obtained in 15 out of 19 cases examined and the average bilirubin content was 0·69 mgm. per 100 c.c. The microscopic examination of a centrifuged deposit of the fluid revealed the presence of a few lymphocytes and endothelial cells in almost all the cases (Table VI, Part I). A guineapig inoculation with the fluid was done in 4 cases with negative results.

TABLE VI—SHOWING FREQUENCY OF PARACENTESIS ABDOMINIS PERFORMED ON 88 CASES OF PORTAL CIRRHOSIS WITH ASCITES

A. *Slightly improved:*			
No paracentesis done	2	4·5 weeks	
Once	2	7	"
Twice	1	3	"
Four times	1	20	"
Five times	1	17	"
	7		
B. *Discharged without improvement:*			
No paracentesis done	15	2 weeks	
Once	16	2	"
Twice	15	4	"
Thrice	8	4·4	"
Four times	6	5·3	"
Five times	2	6	"
Six times	3	3·6	"
Ten times	1	8	"
Thirty-seven times	1	62	"
	67		
C. *Died:*			
No paracentesis	3	1 week	
Once	11	2 weeks	
Twice	3	3	"
Thrice	6	4·3	"
Four times	3	6	"
Five times	2	5	"
Six times	2	7	"
Eight times	1	11	"
Fifteen times	1	25	"
	32		

The Takata-Ara Reaction—This test was performed on 60 cases; it was positive in 51, doubtful in 2 and negative in 7 cases. The significance of the test, especially from the point of view of prognosis, has already been discussed in Part I.

Treatment

The patients genarally came in a fairly advanced stage with a huge accumulation of fluid in the peritoneal cavity. In most of the cases detailed investigations and adoption of particular form of therapy were possible only after they had been relieved by an initial paracentesis. The diet allowed for these patients was the same as described in Part I for cases of nutritional ascites, so also the routine diuretic mixture. Iron in the form of Blaud's pill by mouth and liver extract parenterally was administered where indicated. After an initial paracentesis a mercurial diuretic (neptal 0·5 c.c. intramuscularly) was given in order to prevent further distension of the abdomen.

Under this regime the progress of the majority of the cases, as compared to that of the nutritional ascites, was unsatisfactory, and paracentesis and mercurial diuretics had to be repeated.

Only two cases out of 108 made a clinical recovery in the first instance without requiring paracentesis; out of these one was readmitted with a relapse after about a year and died in hospital four months later; the other returned after a month with ascites in a very low condition and died four days later and a post-mortem examination revealed advanced liver cirrhosis.

Seven cases were discharged clinically improved as judged by improvement in general condition and *prolongation of intervals between paracentesis.* A follow-up of these cases, however, was not possible.

Out of 67 cases discharged without any improvement in the clinical condition, 4 left the hospital in a moribund condition, 2 were re-admitted within a month and died, and 3 were reported to have died one to two months after leaving the hispital. Lastly, 32 cases or 29·6 per cent died in the hospital within 1 to 25 weeks after admission.

As regards the frequency of paracentesis it will be seen from Table VI that this was resorted to four times or more in 23 or 26 per cent of all cases who had to be tapped for the relief of abdominal distension, and *in one case it was done 37 times during a period of 62 weeks.* This finding is in contrast to the view expressed by Rolleston and McNee (1929) that cases of cirrhosis of the liver do not live long enough to require tapping more than two or three times.

Surgical Treatment—The operation of omentopexy was tried in 3 cases without any beneficial effect; one died within a week after the operation and in the other two the fluid in the peritoneum was re-accumulating as usual.

Mode of Termination

The patient gradually becomes weaker and weaker, mentally sluggish and depressed and later semiconscious or completely comatose. The pulse, however, remains good till the final stage. Sometimes these patients become delirious and boisterous only to relapse again into depression terminating in death. Some cases do recover from this crisis to undergo a second or a third attack ending in death. This was the mode of termination in most of the cases. In a certain number, however, death was preceded by an attack of intractable diarrhœa and dehydration.

A post mortem examination was obtained in 8 of the fatal cases and the clinical diagnosis was confirmed. In these cases, there was no evidence of chronic dysenteric ulceration, chronic peritonitis, nephritis or peritoneal tuberculosis.

Aetiology

Causation of Ascites—Mc Indoe (1928) by his experiments on cirrhotic livers, observed that there was a marked diminution in the hepatic vascular bed, that the normal portohepatic venous relationship was lost and that the blood supply of the surviving liver cells was mainly from the hepatic artery. He considered that ascites was associated with a lack of balance between progressive intrahepatic vascular sclerosis and the development of collateral channels. On the other hand, Bollmann (1928) states that when a dog's liver is damaged by ligation of bile ducts no ascites develops so long as the animal is kept on a carbohydrate diet, but ascites develops after a few days of feeding with meat to disappear again when carbohydrate diet is resumed. Bollman (1938) has further shown that in dogs ascites associated with liver injury produced by carbon tetrachloride disappears 2 to 3 months after discontinuation of the drug, concomitant with the improvement in liver function. He has also noted a relationship between serum protiens, particularly albumin, and hepatic damage; the serum proteins were inversely proportional to the degree of liver damage and no ascites was observed when the protein levels were normal. According to Madden and Whipple (1940) all protein is primarily derived from the liver where it has been slowly synthesized. Post and Patek (1942) have noted low plasma proteins with altered albumin globulin ratio in cases of cirrhosis of the liver; further, clinical improvement was associated with a rise in serum albumin content and they conclude that reduction in serum albuñin was an essential factor in the formation of ascites. Ratnoff and Patek (1942) have suggested that the level of serum albumin was the critical factor, whereas portal hypertension decided the site of collection of fluid in the peritoneal cavity. As noted earlier, in the present series the serum proteins were low with albumin fraction less than 3 per cent in all the 8 cases examined. All this together with observations in the present study that some of the patients could live long enough to have repeated paracentesis done (in one case as many as 37 times) and prolongation of the intervals between tappings in some of the favourable cases, as also the disappearance of ascites in at least 2 cases without paracentesis under a regime of rest and liberal diet, would probably show that malnutrition and low plasma protein levels were important, if not the only actors, in the causation of ascites in cases of cirrhosis of the liver, and that the onset of ascites was not necessarily indicative of a rapidly fatal issue.

The average survival period of 28 fatal cases after the onset of ascites was 22·5 weeks. It was not possible to observe the effect of a specially high protein diet as advised by Patek and Post (1941) who have reported a prolongation of the survival period of the patients treated by a nutritious diet supplemented with vitamin B-complex as against the controls.

Diet and Cirrhosis of the Liver—Various views have been held in the past regarding the causation of cirrhosis of the liver in India. Sutherland (1905) stated that out of 163 cases of cirrhosis of the liver 138 had never touched alcohol. He opined that cirrhosis may be secondary to dyspeptic changes induced by spices and condiments. The experiments of Bollman (1938) show that alcohol by itself is not likely to lead to any considerable liver damage, but in combination with other irritants like carbon tetrachloride or a fatty diet or starvation it can rapidly lead to fatty degeneration. Other factors like amœbic dysentery (Rogers, 1911), bacillary dysentery (Menon, 1935), and chronic malaria (Hughes and Srivastava, 1927) have been held as important in the ætiology of liver cirrhosis. Rao (1937) as a result of his studies on cirrhosis of the liver at Vizagapatam has concluded that 'Portal cirrhosis is not due to a single cause but due to several factors of which dietetic errors, syphilis, chronic bacillary dysentery, and splenic anæmia, acting either alone or in combination, appear to be important.'

Experimentally, Rich and Hamilton (1940) claim to have produced hepatic cirrhosis in rabbits fed on a diet deficient in some unknown factor present in yeast; the diets, however, contained a fairly large amount of fat in the form of lard or 'crisco' (hydrogenated cotton-seed oil), but fat is not the natural diet of rabbits. Machella and Maguire (1941) repeated this experiment on albino rats with negative results. Blumberg and McCallum (1941) observed cirrhosis of the liver in rats fed on a high fat diet but this was prevented when choline was administered along with the fatty diet. Lillie *et al* (1941) have reported the occurrence of cirrhosis of the liver in some of the rats placed on a diet low in proteins and sulphur containing amino acids. Rao (1942) has noted necrosis of the liver in some of the monkeys placed on a 'poor South Indian diet,' consisting mainly of rice. In South Africa, Gillman (1944) has observed that when rats were fed on a diet similar to the one consumed by the natives, consisting mostly of maize, for prolonged periods they developed cirrhosis of the liver. Himsworth and Glyn (1944) on the basis of their experiments on rats observe that deficiency of the amino acid, methionine, results in the occurrence of irregular areas of massive necrosis in the liver which later on leads to post-necrotic scarring and hyperplasia of the surviving liver cells (nodular hyperplasia) and that this condition may simulate Laennec's cirrhosis.

As pointed out earlier a history of dietetic errors was frequently obtained in the present series of cases, and further only 40 out of 108 cases took alcohol occasionally in the form of a fermented palm sap, the alcohol content of which is only about 7 per cent and this also contains some yeast. In general, although dietetic deficiencies are common in India particularly among the poorer classes, the conditions, however, differ in different parts. Various dietetic surveys have shown that the diet consumed by an average Punjabi peasant is superior to that of his counter part in South India. But sufficiently accurate data are not available to judge the prevalence of ascites or cirrhosis of the liver in different parts of the country; further, the exact morbid anatomical and histological appearances in the livers of such cases require a careful study in view of the recent experimental observations pertaining to the ætiology of the hepatic cirrhosis. It is however worth noting here that Tirumurti and Rao (1934) have observed nodular hyperplasia in some of the livers examined post mortem at Vizagapatam. In the present study, 52 cases out of 343 or 15 per cent, have been classed as nutritional ascites; the majority of these made a complete clinical recovery and some of them are known to be well for periods as long as two years without recurrence of ascites. It has also been noted that at least 2 cases, who were classed in the first instance as nutritional, came back subsequently with a fully developed picture of cirrhosis of the liver (*vide* Part I). It is, therefore, suggested that ascites in these cases may at first be merely a manifestation of malnutrition without gross hepatic lesions, and capable of recovery, and that a continued state of malnutrition perhaps, associated with other factors like chronic malaria, dysenteries etc., may ultimately result in a fully developed picture of 'decompensated portal cirrhosis' when the chances of complete recovery are remote.

The use of multiple liver function tests as suggested by Trowell (1944), Watson (1944) and Higgins *et al* (1944) including a biopsy examination of the liver tissue (Tripoli and Fader, 1941) is likely to be very helpful in the diagnosis of early cases of cirrhosis of the liver. Further, a regular and systematic follow-up of cases who have either recovered or improved may help in understanding and possibly, modifying the course of the disease favourably.

Summary

1. An analysis of 127 cases of ascites associated with cirrhosis of the liver has shown that 108 were cases of portal cirrhosis, 13 biliary cirrhosis (adults and children) and 6 syphilitic hepatitis.

2. Observations on the 108 cases of portal cirrhosis are presented and discussed. The condition is common among adults and the middle-aged of poor cultivator and labouring class; a history of subsistence on a defective diet is frequent; clinically, these cases are characterised by the presence of ascites, prominent abdominal veins, general emaciation and usually oliguria; hæmorrhagic phenomena such as hæmatemesis, epistaxis and melena were not common in our series. They show a moderate degree of anæmia, low serum proteins with a reverse of the albumin-globulin ratio, a high degree of dye retention by the Rose-Bengal test and a positive Takata-Ara reaction in most of the cases. Response to treatment is not satisfactory and the clinical course is in marked contrast to that of the cases of nutritional ascites described in Part I. A large majority of the cases were discharged without

much improvement in the condition after repeated paracenteses. Nearly 30 per cent of them died in the hospital within 1-25 weeks after admission.

3. The occurrence of oliguria in most of the cases without concomitant signs of renal failure is noted; further investigations are necessary to elucidate the cause of this condition.

4. The causation of ascites in cases of cirrhosis of the liver is discussed and it is suggested that malnutrition and low plasma protein levels are important factors.

5. The literature on the aetiology of cirrhosis of the liver in India and that on the experimental production of hepatic cirrhosis in animals by feeding on defective diets is reviewed.

While defective diet was fairly common, a history of alcohol (and that too in the form of fermented palm sap) was obtained in only 40 cases (37 per cent).

While a comparison of the dietetic deficiency and occurrence of ascites or cirrhosis of the liver in different provinces of India would be interesting, it is felt that sufficiently accurate data for such comparison are not available.

6. It is suggested that cases classed in the present study as 'nutritional ascites' may at first be merely due to simple malnutrition without gross hepatic lesions and capable of recovery, but that a continued state of malnutrition, perhaps, associated with other factors like chronic malaria, dysenteries etc., may ultimately result in a fully developed picture of 'decompensated' portal cirrhosis.

Conclusions

1. In an appreciable percentage (15 per cent) of patients presenting ascites as a prominent part of the clinical picture and seeking admission for its relief the condition is probably attributable to malnutrition. These cases do not show obvious signs of any named disease; their prognosis is good and fairly rapid and lasting recovery takes place on a balanced nutritious diet.

2. A much larger percentage of the ascites cases (37 per cent) are due to cirrhosis of the liver, mostly portal. Their prognosis is unfavourable and response to treatment extremely poor.

3. It is possible that cases of the first kind may develop into the second under continued unfavourable nutritional conditions.

Acknowledgment

This work was done by me working as 'Medical Assistant' in the Ascites Enquiry, under Dr. S. W. Hardikar, Professor of Pharmacology, Osmania Medical College and Honorary Physician, Osmania Hospital, for which the Osmania University and the Indian Research Fund Association had kindly provided special grants.

I am very much grateful to Professor Hardikar for his guidance and advice throughout the course of this investigation and in the preparation of this paper.

I have to thank Dr. H. Hyder Ali Khan, Director, Medical and Public Health Department and his successor, Lieut.-Colonel K. N. Waghray, for giving me the opportunity of engaging in this work; Colonel Waghray has also kindly given permission to publish this paper. To Dr. S. A. Jabbar, Superintendent, Osmania Hospital, I am thankful for sympathetically giving facilities for work in the hospital.

The investigation was rendered possible by the hearty co-operation from the various departments of the Osmania Medical College and the Osmania Hospital. While it is not possible to mention everyone by name, I must express my sincere thanks to Dr. V. G. Borgaonkar, the gynæcologist, for special examination of female cases; to Dr. D. B. Kulkarni, the pathologist and his assistant Dr. M. C. Aiamma for carrying out the Wassermann Reactions and co-operation in other laboratory tests; to Dr. M. Munawar Ali, Surgeon, for advice and surgical treatment in some of the cases; to Dr. S. A. Rahman, Professor of Physiology for some of the electrocardiograms; to Dr. M. Shah Nawaz Khan, Professor of Clinical Medicine for examination of some of the ascitic fluids for tubercle bacilli; to Dr. Ali Hussain, Professor of Pathology and Dr. Yeswant Rao of the Pathology Department for post mortem examination; to Dr. Fazle-Kareem Khan, Reader and Dr. C. D. Nagaratnam, Demonstrator, Pharmacology Department for general help, and to Mr. M. Ghouse Mohiuddin, Chemist, Pharmacology Department, for this valuable help in acquiring the technique of biochemical tests.

References

Bollman, J. L.—*Arch. Path.*, **6**:162-163, 1928.
Idem—*Ann. Int. Med.*, **12**:1-5, 1938.
Blumberg, H. & McCallum, F. G.—*Science*, **93**:598-599, 1941.
Gillman, J.—*Brit. M. J.*, **1**:29, 1944.
Himsworth, H. P. and Glynn, L. E.—*Lancet*, **1**:457, 1944.
Hughes, T. A. and Shrivastava, D. L.—*Indian J. M. Research*, **28**:427, 1927.
Little, R. D., Daft, F. S. and Sebrell, W. H.—Public Health Rep. **56**:1255-58, 1941.
Machella, T. E. and Maguire, E. F.—*Proc. Soc. Exp. Biol. and Med.*, **46**:502-507, 1940.
Madden, S. C. and Whipple, G. H.—*Physiol. Rev.*, **20**:194-215, 1940.
McIndoe, A. H.—*Arch. Path.*, **5**:23-42, 1928.
Menon, T., Bhaskara—*Indian J. M. Research*, **22**:4, 1935.
Mukherjee, H. N. and Gupta, P. C.—*Indian M. Research*, **18**:810, 1940.
Patek Jr. A. J. and Post, J.—*J. Clin. Invest.*, **20**:481-505, 1941.
Post, J. and Patek Jr. A. J.—*Arch. Int. Med.*, **69**:67-90, 1942.
Rao, M. V., Radhakrishna—*Indian J. M. Research*, **21**: 2nd Oct., 1933.
Idem—*J. Indian M. A.*, **6**:195, 1937.
Idem—*Indian J. M. Research*, **30**: 2 April 1942.
Ratnoff, C. D. and Patek, Jr. A. J.—*Medicine*. **21**: 3 Sept., 1942.
Rich, A. R. and Hamilton, J. D.—*Bull. Johns Hopkins Hosp.*, **66**, 1940.
Rogers, L.—*Indian M. Gaz.*, 1911.
Rolleston, H. and Mc Nee, J. W.—Disease of the Liver, Gall-bladder & Bile Ducts. 3rd edition, 1929. Published by MacMillan & Co., Ltd., London.
Rahman, S. A.—*Indian J. M. Research*, **24**:173-179, 1936.
Sutherland, D. W.—Quoted by Rolleston & McNee (1929).
Tirumupti, T. S. and Rao, M. V. Radhakrishna—*Indian M. Gaz.*, **69**:74, 1934.
Wayburn, E. and Guerard, C. R.—*Arch. Int. Med.*, **66**:161-172, 1940.
Weiss, S.—Diseases of Liver, published by Paul B. Hoeber Inc., New York, 1933.

PUBLIC HEALTH AND POST-WAR RECONSTRUCTION

With Special Reference to the United Provinces

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Public health has been defined by Sir Arthur Newsholme as the work embracing all activities which prevent disease and enhance the health of the community and which can be carried out efficiently by communal than by individual efforts. These are the measures which postpone death, prevent damage to health and raise the physiological standard of human life. The public health activities should be directed to cultivate the best physical and mental potentialities of every individual and postpone each death to healthy old age. From this it is obvious that we have to think in terms of positive health of the people than mere sanitation and control of epidemics and the efforts made so far need to be intensified on a much bigger scale.

Present Condition

The death rate and high infant mortality in India with attendant avoidable sickness make one shudder. Economic loss too is terrible. A large proportion of our people are living in lowered health and that this condition is preventible cannot be denied.

TABLE—SHOWING COMPARATIVE BIRTH, DEATH & INFANTILE MORTALITY RATES IN DIFFERENT PROVINCES IN INDIA DURING 1941-1943

Provinces	*Birth rate*			*Death rate*			*Infantile mortality rate*		
	1941	1942	1943	1941	1942	1943	1941	1942	1943
1. U.P.	32·08	28·50	24·71	19·28	18·49	19·23	131·2	143·09	134·57
2. Central Provinces ..	42·47	39·35	37·04	32·01	33·39	27·68	215·28	232·31	206·13
3. Bombay	36·49	34·04	35·42	25·81	24·26	23·31	158·95	160·52	157·39
4. Madras	39·04	33·81	31·12	25·82	22·49	25·25	170·91	159·7	172·29
5. Bengal	29·56	23·93	18·76	21·97	20·19	30·90	155·70	154·32	195·76
6. Bihar	28·91	25·48	20·50	19·48	15·45	16·06	108·18	100·99	98·24
7. Orissa	30·85	31·50	30·75	28·04	25·43	30·50	225·35	190·69	202·55
8. Punjab	39·54	36·71	32·40	24·68	28·33	24·97	185·20	213·63	189·89

Essentials of Public Health

If we can give the people (1) better nutritional status, (2) healthy houses with sanitary surroundings, (3) better education, and (4) a good water supply we would have gone a long way in solving the problem before us.

Poverty—Poverty and ignorance are two great obstacles to public health work. The average income of an Indian is very low. This will have to be raised by every possible means such as greater industrialisation including cottage industries, improved agriculture, better communication etc. etc.

I shall be wandering off my beat if I tried to discuss here the socio-economic conditions of the people but I cannot refrain from referring to these as they have a very important bearing on public health. Col. J. C. De presiding over the Sixth Bengal Provincial Medical Conference at Howrah on 26th November this year declared that the problem of health in backward countries like India was mainly the problem of food and feeding. According to Prof. A. V. Hill disease and malnutrition so worked together in India that any epidemic like that of 1918 might produce a major catastrophe. Food of the needed quality is a big factor in building up a healthy community.

Education—Goethe has said, "Against stupidity even Gods fight in vain". All round primary education with special attention to the girls, along with schools for adults will not only facilitate the task of the health department but will be important steps in creating health conscience. Emancipation of women is a great factor in the march of nation's progress and the subject requires earnest attention.

Here again economic condition will play an important part. The poor cannot afford to send their boys and girls to school. Not only that but these youngsters are made to work to supplement the meagre family income. This vicious circle has to be broken. Public opinion has to be developed. The laws in advance if unsupported by public opinion remain more or less inert. Public health measures cannot be more than partially successful unless they carry with them the conviction of the people. A health worker who argues that he is concerned only with the enforcement of public health measures for preventing infection is condemning himself to relative sterility of efforts.

It must be acknowledged here that public health has been receiving greater attention from the Government for some time but no body will deny that much more remains to be done. These are the days of post-war slogans and it will be some satisfaction if out of the evils of the war a solid public health scheme emerges. I shall briefly indicate the lines on which the scheme might be developed.

Health Centres

The large majority of our population lives in rural areas and any scheme proposed should cater for their needs first. There should be a dispensary for an area of about 30 to 80 sq. miles depending upon the density of population and local conditions, in charge of a qualified doctor. As dispensaries have so far conveyed to us the idea of curative aspect only I would prefer to call these Health Centres. Such a name would create the needed public health basis emphasizing importance of preventive over curative, though not neglecting the latter. These health centres will be

the pioneers of all health activities. To each of these centres a nurse-midwife and a vaccinator *cum* vital statistics clerk should be attached. A compounder to help the doctor and a *dai* to help and accompany the midwife will I think be needed, besides a part time sweeper. Financially it is not a feasible proposition to have anti-malaria squads at all the centres but where malaria incidence is unduly high a squad or squads should be attached. Buildings will have to be improvised and replaced by suitable new buildings in due course. Attached to such a building there should be a shed for detention of serious cases to be transferred to the district health quarters. Some 3000 to 4000 health centres will be required. The existing dispensaries belonging to District Board, Rural Development and other departments can be absorbed in the scheme. As the requisite number of staff will be available only gradually and in a few years' time expenditure will not be much at the beginning and will be stepped up as the new centres are opened.

Duties of the doctor at the health centre—With a limited area to be looked after I would expect the doctor in charge to pay regular visits to the villages in his circle. The distance will not be much and can be covered comfortably on a bicycle. He will be in complete charge of preventive as well as curative sides of medicine in his circle, will render first-aid in epidemics, control vaccination and vital statistics, inspect school children, supervise the work of midwife, and anti-malaria squads if any and so on.

Vital statistics clerk—The present registration of vital statistics has been admittedly very defective and a vaccinator for such an area can do this work also besides vaccination.

Nurse-midwife and dai—A nurse is essential to follow up school children in the families and establish contact with the ladies. Until there is scope both for a nurse as well as a midwife a nurse qualified in midwifery can work. As it is difficult for a female to visit villages alone a *dai* is necessary. Like all schemes this scheme also will be modified from time to time, in the light of experience.

Sanitary inspectors—Instead of increasing the number of sanitary inspectors in the districts I would rather increase the number of centres and the doctors.

Control of epidemics—For control of epidemics the present sanitary staff can be supplemented by reserve mobile units. The need for control of epidemic will be fewer as the health centres succeed in their aim.

It is needless to say that such doctors will be given a short training in public health course. These doctors should be selected for higher posts of the department after they have taken the public health diploma for which facilities should be allowed. This will be an incentive for good work. Thus adults will be contacted by the doctor at the centre and in the villages and the females through the nurse.

School children—The nurse will also approach the children but that will chiefly be the concern of the village school teacher who should work in close co-operation with the medical officer. Arrangement for school meals is urgently called for.

From my experience in the rural area I come to the conclusion that the department should supply material and the villagers labour, for drinking wells.

District Health Officer

There should be a chief medical officer with public health qualifications for each district instead of Civil Surgeons and District Medical Officers of Health. He will be in charge of curative as well as preventive sides with one or more medical officers for the hospital work and one or more touring officers with public health qualifications for supervision of health centres and control of epidemics. There should be hospitals for specialist treatment in surgery, x-rays, eye diseases etc. Tuberculosis and venereal clinics along with a laboratory should be attached to the district hospital and a few beds specially provided for the patients suffering from the former. An infectious diseases hospital is urgently needed not only at the district head quarters but at all the municipalities where there is none at present. In epidemic areas detention sheds should be provided in big villages and attached to the health centres.

An ambulance car at each district head quarters for bringing serious cases from the interior should be provided.

As a logical conclusion to such a scheme medical and public health departments will have to be under one head in the province.

Urban areas naturally have problems of their own. Existing water mains and sewer system have become inadequate and inefficient in many cases. It is beyond the financial abilities of most of the municipal boards to think of relaying these without Government aid. In big municipalities a sanitary engineer or an overseer working in close collaboration with the medical officer of health is called for. Laboratory service for examination of materials from the private practitioners is needed. Incidence of tuberculosis is much higher than the official records indicate and vigorous preventive and curative measures are urgently required, by providing tuberculosis clinics and nurses, means for satisfactory disposal of sputa, tuberculosis hospitals and sanatoria at suitable places. Accommodation for lepers, infirms and mental cases will have to be adequately increased.

Public Health Education

I have noticed that articles on public health written in popular language are read with great interest not only by the doctors but also by the public. This is a good sign and such a craving should be satisfied. I would suggest that such short articles should be invited from the doctors on payment if necessary, and published through the department with such changes as necessary. Many daily, weekly and monthly papers will be glad to receive such authentic information on the subject. Such articles in special official papers have not been a success as far as I know. I have been giving this matter a very careful consideration and even at the expense of being considered dogmatic I have no hesitation in saying that this is a cheap and easy way to approach the public these days. There

(Continued at foot of col. 2, page 307)

SNAKE-BITE

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Introduction

The importance of the subject needs no emphasis. Of all the dangerous animals, the snake is feared and hated the most. The bite is so instantaneous and stealthily given that the victim gets very little chance to defend himself. The interval between bite and death may be only minutes in some cases. Before about 1910 when the anti-venomous serum was evolved, there was no sure remedy for snake-bite.

The whole world loses about one hundred thousand human lives annually, of these about 40,000 are lost by India. This comes to about 100 deaths a day. It is estimated that half the deaths are due to shock alone, and the rest are due to effect of venoms.

The causes of this fatal shock are:—

(1) A wrong notion that all snakes are poisonous and that there is no treatment for snake-bite.

(2) Inability of the public to distinguish at a sight, the poisonous from the non-poisonous varieties.

(3) Misleading ideas, propagated by the snake-charmers, about the snakes, for their own selfish motives. They play upon the credulity of the public and exploit them, for example they tell the public that the rat snake or Dhaman, the earth snakes, and the python are poisonous etc. All these statements are wrong.

The need of the time is to teach the public that—

(1) About 80 per cent of all the snakes met with are non-poisonous, therefore most of the bites are from non-poisonous snakes.

(2) The species that are responsible for majority of the deaths are the common Indian cobra and the Russell's viper.

(3) The antivenene, or the antivenomous serum, prepared by the Central Research Institute of Kasauli, and Messrs. Drug House (India) Ltd., 52, Sambhunath Pandit Street, Calcutta, is polyvalent and fully effective against the common Indian cobra, the Russell's viper and partly against the Echis.

This is the only drug against snake-bite, known to science. All the other so called snake-bite-cures have proved utterly useless so far. About 100 indigenous snake-bite cures were tried at the Haffkine Institute of Bombay by Dr. Mhasker and Rev. Father Caius in 1931. None proved of any avail. I have tried about a dozen, with similar results.

(4) Serum treatment is required in the case of venomous bites only. Non-venomous bites need nothing more than a clean dressing and an assurance to the victim, that the snake is non-poisonous and nothing will happen.

(5) It is essential for everybody to learn how to distinguish the poisonous from the non-poisonous. This can be done by studying scale-arrangement from Col. Wall's chart, colour markings from Col. Knowles' chart and remaining characters from a collection like that of mine.

This lecture gives a brief sketch of the problem. Pictures could not be included in it, which will appear in a book to be published later. Lt. Col. Gharpurey's book, 'The Snakes of India' published by The Popular Book Depot, Lamington Road, Bombay, is a good guide so far available in the market.

(6) To propagate this useful knowledge among the public it is advisable that the subject should necessarily be taught in all medical colleges and schools. All hospitals should make a collection of local varieties of snakes by offering a reward for each snake. Such collections should be kept as exhibits in all public places, such as museums, schools, courts, railway stations, police stations, parks etc.

(7) The knowledge about the subject should be an integral part of all educational programmes.

Position of the Subject

(a) *Work done in the past*—About the year 1868 the Government of Bombay started a campaign to eradicate the echis from the Ratnagiri District by fixing a reward of two annas per snake. More than one hundred thousand snakes were caught in one week. The reward was reduced to one anna, the amount sanctioned finished but not the snake. Many deaths are caused by Echis in that area.

In 1870, Sir J. Fayer, under the patronage of the then Viceroy made a thorough study of the subject, carried out biting experiments on animal, collected authenticated cases of snake bite and left behind him a monumental work 'The Thanatophidia of India'.

In 1878 potassium permanganate was tried as an antidote.

About 1886 a commission was appointed by the Government of India to find out the interval between infliction of a bite and stoppage of respiration in cobra-bitten subjects.

From 1905 to 1919 Col. F. Well made an extensive study of the animal. The present position of the subject is due to his labours. During the same time antisnake-bite serum was evolved.

In 1912, W. Bannermann, proved that potassium permanganate was useless as an antidote.

In 1913, 1914, 1915 Acton and Knowles studied the subject in a thorough manner, established several important facts and collected very reliable data.

In 1931 Dr. Mhaskar and Rev. F. Caius tried about 500 so-called indigenous snake-bite cures and proved that none was of any avail.

(b) *Work done by the author*—Finding myself ignorant of the identity of snakes that I met, and unable to diagnose cases of snake-bite to my satisfaction, I started a study of the subject, for which this collection of mine was a necessity. I have been able to collect 18 species, of these 4 are poisonous, 14 non-poisonous,

collection continues in the hope of getting new species and better specimens of the existing species. Without a collection like this, one cannot grasp the details of the subject. I have been trying antisnake-bite cures sent to me for opinion by the public. A study of the action of cobra venom is being done at present on animals.

(c) *Work to be done in future*—Collections of specimens of snakes should be made, as suggested above in every town. These collections should be placed at places of public importance, accompanied by natural colour pictures, line drawings, models, descriptive cards and directions for distinguishing between poisonous and non-poisonous varieties and treatment of snake-bitten subjects.

Knowledge about the animal should be widely spread among the public by the public health and propaganda departments. Knowledge removes superstition and cowardice. Spread of this knowledge will eliminate deaths occurring due to shock.

Every medical man is conversant with difficulties connected with the use of antisera, especially when given intravenously. Serum sickness coming on about a week after the injection is a common nuisance to be tackled. Advent of serum shock may complicate matters seriously in hypersensitive individuals. If we can find out a substitute for the antivenomous serum it will circumvent these difficulties attending the use of serum. Serum cannot be administered by a layman. In areas where these accidents occur a medical man is difficult to get ordinarily.

The present serum is partly effective against the Echis and not at all effective against the common krait, the banded krait, and the king cobra. These latter varieties cause a certain number of deaths which cannot be prevented in the present state of our knowledge. Science has still got to solve this problem. We are in need of a remedy, which may be very easy to administer, so that every one, even if he is a lay person may be able to administer the remedy to the victim or the victim may be able to take it, because most of the snake-bite accidents occur in out-of-the-way places, where a medical man is not easily available. Snake venoms act very fast, therefore in many cases by the time the victim reaches a medical man, poisoning is far advanced and chances of recovery are lessened. A study of the animal on a bigger scale may reveal a method of exterminating the animal. We may be able to find out a parasite for the snake, which if introduced into our country may finish this dangerous reptile. Some years ago, a gentleman brought a variety of cochineal (an insect) to Hyderabad. It spread and finished every cactus plant. The cactus had been a great nuisance, and there is no trace left now. In the same way a study of the enemies of the snake is worth the trouble and expense.

Mr. Fitzsimons of Port Elizabeth (South Africa) suggests keeping rat-eating snakes in houses to get rid of rats. We should find out some snake-eating animal and introduce it into our land.

I got lot of snakes from the local drainage disposal works. One day I asked the man who brought these animals, how he could easily get so many. He said 'Insects collect in the light near the electric posts. Frogs come to eat these insects. Snakes come to eat these frogs. I go and kill the snakes'. Animal life means parasitism.

There is a bird of prey, in United States of America, called the secretary bird. It feeds on snakes. Webster's dictionary gives an illustration of a fight between a secretary and a cobra.

A study of the enemies of the snake is a very prospective subject. Universities should create research scholarships for the purpose. I am sure the efforts will not go in vain.

Research work on the action of the venoms of the animals must continue till the remedies for all the snakes are discovered. I know even highly educated and highly placed people expressing an opinion that because a serum is available, further research on snake-bite is not necessary although the fact remains that the present serum fails in treatment of cases bitten by the common krait, the banded krait, the king cobra, the coral snake, and the echis. Of these species the common krait, the king cobra and the echis cause a good number of deaths.

About such men I would say, may God put their brain on the right path. By giving this opinion they thwart the attempt to find out an antidote against the venoms of these deadly reptiles. Our armies fighting in Burma at present are facing these snakes, because king cobra and the banded krait are very common in Burma.

Brief Anatomy and Physiology of the Snake

The snake belongs to the class reptilia. It has no limbs. Its body is covered with scales. An animal not covered with scales is not a snake. The eyes of the snake have no lids. The body of the snake is divided into a head, a body, and a tail. Only in some snakes the area behind the head is narrow and can be called a neck. The junction of the body and tail is marked ventrally by the cloaca or the vent. The gape of the mouth extends far back. The bones forming the upper jaw and the head are moveable. The mandible is divided into two parts anteriorly. These two parts are united by an elastic ligament. All this arrangement is responsible for the ability of the snake to open its mouth very wide in both vertical and horizontal planes and engulf animals bigger in size than itself.

Eyes are not provided with lids. The cornea is one scale and is shed along with the skin. The pupil is circular in colubrids, vertical in the vipers and horizontal in tree-snakes. Eye-sight is good and snakes are nocturnal animals.

The hearing apparatus is lowly developed. External and middle ears are wanting. Internal ear is present. Therefore the snake cannot hear sounds conducted through air, but can hear sounds conducted through earth. It cannot hear clapping of hands but can hear a thump on the ground, the vibrations reaching the internal ear through the earth and snake's body and bones.

Tongue is bifid. It is only an organ of feel and not of swallowing. Snakes do not chew their food,

they swallow it whole. Digestion is very powerful. A python swallows a whole pig, and we cannot get, from its excreta, anything except some pieces of hoofs and a few hairs. Venom-glands are modified salivary glands. Venom kills the prey and also digests it. All internal organs are elongated. The vent is a cloaca, and situated at the junction of the body and the tail. The tail is a prehensile organ.

Classification of Snakes

For practical purposes snakes can be divided as follows:—

1. Blind snakes, thin and thick.
2. Constrictors, conical tailed earth-snakes, thick tailed earth snakes, and the python.
3. Vipers. (*i*) Pit-vipers, not found in Hyderabad-Deccan. (*ii*) Pitless vipers, found in Hyderabad-Deccan. These include Russell's viper or chained viper (having 3 chains of spots on the back), and the echis or afai.
4. Colubers. Colubrids, or Colubrines have large scales or shields on their caput. This is a very big group containing poisonous as well as non-poisonous varieties, cobras, kraits and coral snakes are poisonous members, rest being all non-poisonous.

A List and a Short Description of Snakes Found in Hyderabad-Deccan

1. Small blunt-headed blind snake, non-poisonous, Typhlops brahminus—length never more than 6″, about ⅛th″ thick, body shining, covered with small scales, no neck region can be distinguished, dorsum dark brown, abdomen creamy. No belly-plates.

2. Pointed-headed blind snake, non-poisonous, Typhlons acutus—length upto 1 foot, breadth about 1/3″, body shining, covered with small scales, no neck region can be distinguished, dorsum dark brown, abdomen creamy. No belly-plates.

3. Conical-tailed earth-snake, non-poisonous, Eryx conicus—tail about 1½″ long, dorsum covered with irregular dark coloured patches, surface of back rough due to lines made by longitudinal ridges on scales, a longish head, neck region clear, abdomen cream-coloured. Incomplete belly-plates.

4. Two-mouthed earth-snake, non-poisonous, Eryx Johnii—a very dull animal, always coiled up, hiding its head under a loop of its body, head and tail equal in thickness, eyes very small, colour of dorsum dark brown. Back is smooth and shining. Tail less than 2 inches in length, and blunt. Belly is light brown smooth and shining. Incomplete belly-plates.

5. Python (Python Morulus) non-poisonous—a dull heavy-bodied well known snake, head longish, neck region present, the whole dorsum covered by irregular dark brown or black patches, more or less quadrangular in shape, area between the patches brown or yellowish, back is smooth and shining. Tail is short as compared to body length and ends bluntly. The belly is smooth, shining and yellowish. Incomplete belly-plates.

6. Chained Viper, Russell's Viper, Vipera Russellii Vel Elegans, daboia, poisonous—it is a very common snake. Its head is very big, and triangular. There is a white V shaped mark on the head, with apex of the V pointing forward. Its nostrils are the biggest among the Indian snakes. Its pupil is golden coloured and vertical. The neck is well-marked. The whole back is covered by three chains of oblong spots from head to tip of tail. The spots of the central chain are oblong pointed and complete, while those of the lateral ones may be complete or incomplete. The colour of the spots is black or any shade of brown. The back is not shining and is rough due to the presence of a longitudinal ridge on each scale. The tail is short. The belly is shining and cream coloured and the ends of the belly plates are covered by small brownish spots. This snake does not try to glide away when a human goes near, as the cobra does. Its movements are sluggish, but it darts like lightning when attacking its victim. Belly-plates complete. Head covered by small scales.

7. Afai (Urdu), Phoorsa (Marathi), poisonous, Echis Carinata—a very agile, aggressive snake, generally sitting in a figure of 8 like posture, brown in colour. The head is triangular and is covered with a bird's-foot-print mark or a cross mark. There is a neck present. Each flank carries a continuous white wavy line. Between the upper curves of the two wavy lines, are situated rhomboid areas of a darker hue. The back is rough due to the scales being keeled. The tail is short. It is a snake of the sandy areas. Belly-plates complete. Head covered by small scales.

8. The Common Indian Cobra, (Naja Naja Vel tripudans) poisonous—it is a graceful animal. If seen during life, its hood is most characteristic. The body is graceful, of a uniform colour, and does not bear any marking. It is so at least in the Deccan. The cobras from Bengal show some transverse whitish or yellowish stripes on the back, as illustrated by Fayrer in his book 'Thanatophidia of India'. The head is as broad as the body. When in a dead condition, the skin of the neck region can be stretched out, and the spectacle-mark, double or single, if present can be seen. The part of the mark that connects the glass marks, that is the bridge, is convex posteriorly. The area of the neck that surrounds the spectacle mark is more darkly coloured than the rest of the back. It is often speckled with small golden spots. Posteriorly this area is very sharply defined, and extends as far as from 5th to 7th scale as counted in the middle line from the convexity of this bridge. The posterior boundary of the area is sharply defined and carries small black dots that become visible on stretching the skin. I have found this dark area in all the cobras I examined, my number being about 200. Ventrally and just in front of each glass-mark there is present a big pitchblack spot of an irregularly round shape. In some specimens there are extra small dots near this big one. Between the 2nd and the 37th ventral plates, we find 2 or more sets of fully dark-coloured plates. In some specimens I have found all the remaining ventral plates also partially dark coloured. The presence of these dark-coloured belly plates is a non-failing character of the common

Indian cobra. The colour of the belly may be creamy, reddish or slaty. Complete belly-plates. Head covered by shields. Only one scale between eye and nostril scale.

9. The common Krait, Bungarus Coeruleus, Chitti of Bengal, poisonous and fatal—The ground colour of the body is generally shining steel-black, brown specimens are also found. The back is crossed by fine transverse complete, single or double white arches. These arches begin at some distance from the head and extend up to the tip of tail. The colour of the belly is creamy white. Belly plates complete. A line of enlarged hexagonal scales down the middle of the back. Only one scale between eye and nasal scale.

10. Dhaman (Ptyas Mucosus, Zamenis Mucosus), non-poisonous—the common rat-snake. It is very active, and agile animal, attacks ferociously, runs very fast, is generally ground-coloured. The vertical margins of the lower-lip scales are black. On the posterior half or two thirds of the back there are present, transversely placed dark stripes. These stripes are never straight, they are always zigzag. Many of these bifurcate on either flank. Belly-plates complete. More than one scale between eye and nasal scale and so is the case with the remaining eight snakes.

11. The House-snake, Lycodon aulicus, the common wolf-snake, non-poisonous—in most specimens, specially the young ones, there is a white or yellowish collar band round the neck. The back carries whitish or yellowish transverse thick bands. These bands are small and thick and not full arches as in the krait. These stripes start from near the head and end at some distance from the tail, in the krait they start some distance from the head and extend to the tip of tail. The ground colour of the body is always brown. It is never steel-black like most of the kraits. Belly-plates complete.

12. The common filleted snake, the Kukri snake (Simotes arnensis), non-poisonous—this snake grows to about 2 feet. Its body is smooth and brownish in colour. Its back is crossed by deep black cross-bars. The belly is usually white, but may be spotted black. Three marks on the head. These are all V shaped and point forwards. It is so-called because the back-teeth of its jaw resemble in appearance a Gurkha's kurki. Belly-plates complete.

13. The Trinket snake (Coluber Helena), non-poisonous—the body of this snake is slender and smooth. There are black cross-bands on its back. Each black band encloses three or more white islets, which look like small trinkets. In the adult specimens these islets extend in the whole length of the body, while in the young ones, these are present only in the fore part, while the hinder part is free from them, but there is a dark line on each flank. On the neck there are two black stripes extending to the throat. Belly-plates complete.

14. The Chequered Keel Backs or the water-snakes, non-poisonous—all of them have a very distinctive chequered appearance of their back. They are found near ponds. They are very aggressive and very good swimmers.

The ground-colour of the back is greyish or grassy green. The pattern is always of a checquer. There may be lines of alternating black spots, or of white or pink spots. The belly is cream coloured. There are 2 black stripes going backwards and downwards from behind the eye and ending near the angle of mouth and the neck respectively. Belly-plates complete.

15. The Buff Stripped Keel Back (Tropidonotus Stolatus) non-poisonous—it is a small, slender, elongate and graceful snake with a long tapering tail. It is greenish brown and bears two buff or yellow parallel stripes along the back extending from the neck to the end of the tail. There are a number of black cross bars on the black, broken by these yellow lines which are more marked towards the head. The back is rough due to the scales being keeled. Belly-plates complete.

16. Common Green Whip snake (Dryophis Mycterizans), non-poisonous—it is of a bright green colour with an elongate body. Its snout is long and ends in an elongated fleshy upturned appendage, hence its head is characteristic. The tail is sometimes more than one third of the total length of the snake and is relatively longer than in any other Indian snake. The colour of the belly is green, but of lighter shade. It has on each side a narrow white or bluish stripe. Belly-plates complete.

17. The Common Brown tree snake or Bamma snake, non-poisonous, the Common Indian cat-snake (Dipsadomorphus Trigonatus)—the body is slender and elongate, but thick in the middle. The back is of a light yellowish brown or sandy colour with a series of white marks on it, resembling the Greek letter gamma. The head has brownish or yellowish markings.

18. Indian Broze-backed tree snake (Dendrilaphis Tristis), non-poisonous—it is a graceful snake. It grows to 4 feet. Its tail is very long, and is one-third of the whole body length. The back is of bronze colour. There is usually a dark lateral stripe on the back extending from the head to the root of the tail. The belly is yellow and ridged. Belly-plates complete.

From the above description, it will be clear that (1) the external characters of a snake are enough to determine its entity and (2) those of any one species cannot be mixed with those of any other. Because these characters are constant and invariable, hence they can be a reliable guide to recognize a given specimen.

If one can remember the appearances of only the poisonous kinds of one's area, which are never more than a few, the difficulty is solved.

Importance of Subcaudal Scales in Distinction

The lower surface of the tail is covered by scales arranged in one line or in two lines. Krait and echis are the only two snakes, in which the subcaudal scales are arranged in a single row. In the king cobra, the scales near the anus are undivided while the rest are divided.

Colour-Markings as a Guide for Distinction

It is true that the arrangement of scales on the body of a snake is a non-failing guide in determining its identity. From my teaching experience I can say

TABLE OF DISTINCTION BETWEEN POISONOUS AND NON-POISONOUS SNAKES BASED UPON SCALE ARRANGEMENT

SNAKES

Tail compressed sideways and flat		Tail cylindrical and not compressed sideways
Sea-snake, poisonous		Land-snake
Whole belly-breadth covered by small scales. No transverse belly plates present. Burrowing snakes.	Belly plates not broad enough to be extended right across it. Cover only middle ⅛ or ¼ of belly breadth, lines of small scales on both sides of belly plates, visible when animal is on its back. Pythons and earth snakes.	Broad plates covering the entire width of the belly.
Non-poisonous blind snakes	Non-poisonous	
Small scales on the head. No pit between eye and nostril scale.	Small scales or shields on the head and a loreal pit (between nose and eye).	Shields on the head and no loreal pit between eye and nostril.
Pitless vipers-poisonous, fatal.	Pit-vipers poisonous, not fatal	
1. 3rd upper-lip shield touches the eye and nasal-shield.	2. Scales in the central row on the back are enlarged and there may be bands or halfrings across the back. Subcaudal scales undivided.	3. None of the characteristics mentioned in either No. 1 or 2.
Cobra and coral snake. Poisonous and fatal. Neck with hood and with coral spots markings on belly.	Kraits, poisonous and fatal.	Non-poisonous.

N.B.—This table should be studied along with Col. Wall's Chart.

that some people cannot make use of scales for different reasons. There are people who cannot overcome the hatred and loathsome feeling that they have towards a snake. They will not examine it from a near distance. There are others who can never understand it. For these groups, the external appearance is the only guide available. Mr. Prater, Curator of the Museum of the Bombay Natural History Society is against making the external appearance a guide for distinction. He showed me specimens of the common krait in which the transverse stripes started near the head, instead of starting at a distance from it. The latter being a characteristic of the lycodon or the house snake which is harmless. Such specimens of the krait are rare. I never saw one. Moreover the presence of a single row of scales below the tail of a krait and that of two rows below that of a house-snake is enough to decide.

In Bengal I saw a cobra, which has an irregular transverse striation on its back, although the cobra of the remaining Indian provinces shows no such striation.

The external appearance may not serve as an all-India guide, but it can easily be a provincial guide. The term 'external appearance' includes, shape of the head, presence or absence of neck, shape of the body, shape of the tail, proportion between the sizes of these parts, general colour, colour markings, longitudinal stripes, transverse stripes, spots, regularity or irregularity in the shape of the spots etc.

If you look at the specimens of the different species carefully, you will observe that each species has been given by nature a definite external appearance of its own, which is not shared by any other. If several collections like mine are made, and public is given multiple chances of seeing these, they will get acquainted better and the problem will get easier.

(To be continued)

(Continued from page 302)

are not many doctors who though competent in their own lines can write. To remove this handicap I would go even to the length of saying that much of the chemistry taught in D.P.H. course can be usefully replaced by a short course in medical journalism.

Rural development department has radio sets for propaganda work. This needs to be further expanded.

Short public health films should be demonstrated at each cinema show.

The use of ambulance at the district head quarters as publicity van with models etc., in rural areas should be considered. Where the van would not go a bullock cart similarly equipped and moving from village to village for some two months in winter has proved useful in some districts.

Question on hygiene in examination from primary class III upward should be compulsory.

CONCLUSIONS

As already noted I have only indicated the lines and touched the salient points on which reorganisation might be planned. Details are lacking, many gaps have to be filled and finances are to be worked out. But this can be done. My purpose is to stimulate discussion and with your help arrive at an agreed plan. Then it will be time to work out the various details. The scheme particularly refers to U. P. but with suitable modification should be applicable to other provinces also.

There are certain things like compulsory inoculation of *mela* pilgrims and other legislative measures which I would prefer to postpone to the second five year plan by which time the public opinion should have advanced enough.*

* Read at the Scientific Section, XXI All-India Medical Conference, Cawnpore, December 1944.

SPECIAL ARTICLE

SOME ADVANCES IN MEDICAL TREATMENT*

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For the last ten years the progress in therapeutics was so amazing that a text book of medical treatment, published in 1935, to-day reads like a tale of days long by-gone. The milestones of so-called "modern" therapy such as the introduction of salvarsan in 1910, of novasurol, the first injectable mercurial diuretic, in 1920 (Saxl and Heilig), of insulin in 1921 (Banting and Best), of liver therapy in 1926 (Minot and Murphy), the first crystallized hormone in 1929 (Doisy *et al.*, Butenandt), the tremendous work on vitamins which started with Hopkins' brilliant conception of accessory food stuffs in 1906 and Funk's surprisingly correct prediction which diseases would prove to be due to "vitamine" deficiency—all this seems somewhat remote and somehow faded, compared with the spectacular successes achieved since Domagk, in 1935, introduced into therapy sulphanilamide, the first sulpha drug, the Oxford workers rescued penicillin from oblivion in 1940, and the first sample of Swiss Gesarol, which made and makes history under the name D. D. T., reached the U.S.A. in 1942.

SULPHONAMIDES

Starting this review with the sulpha drugs, the most important feature of recent developments seems to be the great simplification in selecting a particular one of them for a certain pathological condition. A year or two ago, one had to stick to fixed indications for sulphanilamide, sulphapyridine, sulphathiazole, sulphaguanidine, sulphadiazine, sulphamerazine etc. To-day, a sufficient amount of sulphadiazine cover all the medical indications. Pneumococci, meningococci, E. coli., our cases of bacillary dysentery and a good many of hæmolytic streptococci, causing the bulk of puerperal septicæmias and tonsillar infections, respond to sulphadiazine better and with less untoward by-effects than to any other sulpha-compound.

The surprisingly good effect of smallest doses of sulphadiazine in *preventing infections* with haemolytic streptococci was proved by mass experiments, involving some 900,000 people, carried out in the U.S. Army and Navy. Carter (1945) reported the following results from the continuous prophylactic use of 1 gm. sulphadiazine per day; the incidence of scarlet fever fell from between 63 and 171 per thousand to zero, of tonsillitis from 426 to 46 and of rheumatic fever from 87 per thousand to zero. He estimates that this procedure saved from 50,000,000 to 100,000,000 dollars. Various authors stress the point that none of the streptococci became sulpha-resistant in spite of those small doses; other investigations (Siegel *et al.*, 1945), however, showed that sulpha-resistant pneumococci may replace those strains which are wiped out by sulphadiazine. It may be mentioned that for the treatment of common colds such as rhinitis, pharyngitis etc., sulpha drugs are not required whereas the high febrile "flu" which occurred here so frequently during the last cold season undoubtedly was cut short by sulphadiazine, usually becoming afebrile on the second day of treatment; in clinically indistinguishable cases, treated with aspirin and diaphoretic mixture, the fever lasted about five days.

Of still much greater importance is sulphadiazine prophylaxis of meningococcic infections; the spreading of such epidemics could be inhibited almost immediately when this procedure started. Meningococcic meningitis still is the undisputed domain of sulphadiazine therapy which, in sufficient doses, proves superior to penicillin, as the latter has to be injected daily intrathecally in addition to the usual intramuscular or intravenous route; in staphylococcus meningitis, however, and in infections of the meninges due to the pneumococcus and haemolytic streptococcus, a combination of penicillin and sulphadiazine seems to be better than either alone.

It is generally recognized that sulphadiazine is preferable to sulphapyridine in the treatment of pneumonias and by far superior to every other sulpha drug in meningococcic infections. But its occasional superiority over sulphathiazole in controlling urinary tract infections is not so well known. In several cases of thiazole-refractory pyelitis in females, in cystitis due to urinary retention in prostatic enlargement or inflammation, we switched over from thiazole to diazine with most gratifying results.

Numerous reports in literature and our own considerable experience show that there is no practical reason for using sulphaguanidine in the treatment of bacillary dysentery. It requires vast doses which trebles the cost, and the main argument for its use that it is not toxic because it is not absorbed through the intestinal wall was proved to be wrong. Sulpha-pyridine, -thiazole and -diazine in very much smaller doses have an equally good effect in a shorter time; and -diazine in our cases never caused vomiting or kidney damage so that we prefer it to anything else. A great additional advantage over -guanidine is that sulpha-pyridine, -thiazole and -diazine can be injected intravenously which obviates all the difficulties of oral administration in vomiting patients. There is no reason why diazine should not be equally as effective in cholera as it is in bacillary dysentery.

PENICILLIN

On penicillin so many investigations have been conducted in streamlined fashion that in a few years an enormous amount of facts have been collected. Very little remains mysterious about the miracle product of *penicillium notatum* (Fleming, 1929), the tremendous *practical* importance of which was discovered in 1940 by Chain, Florey, Gardner, Heatley, Jennings, Orr-Ewing and Sanders, the original Oxford team. The effect of penicillin or Gram-positive bacilli and Gram-negative cocci, such as most streptococci with the exception of *Streptococcus faecalis*, most staphylococci with the exception of some strains of staphylococcus albus, pneumococci, gono- and meningococci, on clostridium tetani, botulinum and perfringens, treponema pallidum and many others, borders on the

* Presidential Address, 2nd Annual Function, Jaipur Branch, I.M.A., March 2, 1946.

incredible, especially, as it is free from any toxic by-effect, and was duly recognized by the award of the Nobel prize for 1944 to Sir Alexander Fleming, Sir Howard Florey and Dr. Ernst Boris Chain.

Absolute indications for using penicillin are subacute bacterial endocarditis, staphylococcal septicaemia, sinus thrombosis, diabetic carbuncle, sulpha drug resistant pneumonia and gonorrhoea; it is very useful in most empyemas and some cases of lung abscess, in "trench mouth" or Vincent's angina (Shallenberger *et al.*, 1945) and in tropical ulcer. It adds considerably to the effect of antitoxin and surgical management, which remains the decisive factor, in gas gangrene. It revolutionised prognosis and treatment of mastoiditis and acute osteomyelitis. But one of its greatest triumphs penicillin achieves is in primary and secondary syphilis, as far as present observations go. Late cutaneous and osseous syphilis respond well to penicillin; the results in neurosyphilis are not striking and cardiovascular manifestations are a contraindication.

The amount required varies from 100,000 units or even less for 24 hours in gonorrhœa, 100,000 to 200,000 units a day for 1 to 3 weeks in S. aureus infections, to at least 300,000 daily units for at least 3 weeks, better for 6 to 8 weeks, in subacute bacterial endocarditis (Keefer, 1945); the number of injections from one to five in gonorrhœa and 70 in syphilis, to many hundreds in some of the chronic infections. That 98 per cent of all cases of gonorrhœa could be cured in 12 hours and almost every case of primary and secondary syphilis in 8 to 9 days by injecting 2,400,000 units, is almost a pity, the fear of infection which is an effective substitute of morals being abolished by the easy cure, with no toxic by-effects to be expected. At present the necessity of many injections still is a deterrent but the reports of successful application of *penicillin by mouth* are increasing in number (Finland *et al.*, 1945, Bunn *et al.*, 1945); 100,000 units given every 3 hours for about 48 hours in formaldehyde-alcohol hardened double capsules, preceded by aluminium hydroxide, gave most promising results in gonorrhœa, cellulitis and pneumonia (Ross and McLendon, 1945). If such attempts to make oral penicillin equally as effective as injected one, finally succeed without too much increasing the expenses of treatment, the complicated methods of prolonging the effect of every single injection to obviate the necessity of injecting every 3 to 4 hours, might become less important.

The permanent limitations of penicillin are its ineffectiveness against Gram-negative bacteria of which the most important are the dysentery group, E. typhosa and the coliform bacilli, against rheumatic fever, tuberculosis, malaria and virus diseases. But even in *amoebic dysentery* where a penicillin effect would be least expected, it proved life saving by eliminating mixed infection. Hargreaves (1945) reported desperate cases of amoebiasis which after 1,000,000 units of penicillin improved miraculously and now responded to anti-amoebic treatment while they proved resistant to every amoebicide prior to penicillin administration.

Streptomycin

A perfect complement to penicillin, apparently, was found in streptomycin by Schatz, Bugie and Waksman (1944). This team discovered that actinomyces griseus, a soil fungus, produces miracle drug number two, which seems to be a most powerful agent against those Gram-negative and acid-fast bacteria which are resistant against penicillin. Helmholz (1945), who a few years ago introduced ketogenic diet into the treatment of *urinary infections*, now reports that streptomycin eliminates completely the most stubborn urinary infectants, streptococcus faecalis and pseudomonas aeruginosa, which resisted every other drug. Results of *typhoid* treatment, though far from perfect, seem promising (Reimann and Elias, 1945). But most interesting are the findings of Feldman and his colleagues (1945) who saw striking effects of streptomycin in experimental *tuberculosis* of guinea pigs, and those of Smith and McClosky (1945) who reported that streptomycin multiplies the effect of promin in this condition. If these results are clinically confirmed, the attack on tuberculosis, the still undefeated enemy of mankind, will be started from two different sides: the sulphone-compounds which seemed until quite recently to have the best chances to conquer acid-fast bacteria, and the new anti-biotic, the greatest draw-back of which is the extreme difficulty in obtaining sufficient quantities. The four biggest American manufacturers together produce hardly one per cent of the present requirement.

Bacitracin

In passing it may be mentioned that a Columbia University group (Johnson *et al.* 1945) found yet another antibiotic, produced by some strains of bacillus subtilis, which they called bacitracin; it is active against much the same organisms as is penicillin, but can be produced in much larger quantities so that it may become cheaper than penicillin, once production starts on a commercial scale.

After this sketchy outline of the properties of new drugs, some of which are still in the experimental stage and not available to us for some time to come, it may be desirable to see whether new applications of older drugs have been worked out, practically important enough to be included here.

Prostigmine

Progstigmine or neostigmine, closely related to the vagus stimulant physostigmine, was used first as the most powerful agent in paralytic ileus and atonia of the bladder. Then, Walker (1934) discovered that it has an astonishing effect on myasthenia gravis, which for a few hours after every prostigmine administration, by injection or orally, becomes normal for all practical purposes. Prostigmine produces this effect by inhibiting cholinesterase (Kennedy and Wolf, 1937, 1938), an inhibition which facilitates the effect of the "vagus stuff" on the neuromuscular junction. On this pharmacological action, which, by the way, has its exact counterpart in the effect of quinine in myotonia (Kennedy and Wolf, *loc. cit.*), Kabat (1945) based clinical experiments with prostigmine in various

conditions of *impaired neuromuscular function;* he reported encouraging results in hemiplegia, facial paralysis, cerebral palsy, rheumatoid arthritis and subacromial bursitis. We tried this treatment in several cases of rheumatoid arthritis and a few hemiplegias and, at least, our patients were not the worse for it.

Another new indication of prostigmine is *paroxysmal auricular tachycardia.* We used it in two cases where every other known therapy had failed and achieved dramatic success, certainly due to the powerful vagus stimulation.

Nicotinic Acid

Nicotonic acid or niacin, a member of vitamin B_2 complex, was found to be one of the most important among the long sought for anti-pellagra factors (Spies, 1938); its administration cures most of the pellagra manifestations. Based on this experience, it seemed tempting to try whether isolated features of the pellagra syndrome, unrelated to pellagra, would respond to nicotinic acid administration. Cleckly and Jolliffe introduced it, therefore, into the therapy of *mental disorders,* an addition to our very limited armamentarium which we found most powerful, especially when amentia, depressive or maniac psychoses were preceded by a febrile disease or appeared during its course. I made intravenous niacin-amide injections an essential part of our routine in treating *delirious cases* of typhoid, pneumonia, cerebral malaria and all the mental derangements following these diseases; the results frequently are highly satisfactory.

Pyridoxine

Since vitamin B_6 (pyridoxine) became available, we are combining six tablets of it per day with nicotinic acid injections, pyridoxine being one of the three pellagra curing factors. This vitamin, quite recently, was found to cure agranulocytic angina when intravenously injected in doses of 200 mgm. per day (Cantor and Scott, 1945).

Equally satisfactory effects of nicotinic acid I reported in *another non-pellagrine syndrome,* presenting some pellgra like features, *i.e.,* entero-colitis preceded or accompanied by stomatitis or glossitis (Heilig, 1943); this condition is of frequent occurrence, especially in South India, and the life saving specific action on it of nicotinic acid was confirmed by Aykroyd (1945).

Liver Diseases

Much work has been done recently on the treatment of liver diseases, based on experiments in animals, which seem to show that fatty infiltration which leads to a cirrhosis of the Laennec type could be prevented by feeding of methionine. This essential amino-acid provides the methyl groups for the manufacture of choline, an indispensable precursor of lecithin which is required to transport the fat away from the liver. Liver necrosis, on the other hand, which is produced by low protein diet cannot be prevented by choline, whereas methionine, cystine, cysteine exert a protective action. These three amino acids have the sulphydril radicle in common. Methionine, therefore, seems to hold a key position in the life of the liver cell, supplying the labile methyl groups which prevent fatty infiltration and cirrhosis as well as the sulphydril radicle which protects against necrosis.

Unfortunately, the clinical application of these laboratory findings was not successful, whereas research based on clinical observation and analysis of dietary habits all over the world, resulted in new methods of *dietary treatment* of cirrhosis of the liver which are highly promising. Patek (1937, 1943) put his cirrhosis cases on a diet rich in protein and ample in carbohydrate and fat; it consists largely of meat, milk, eggs, fruit and green vegetables, supplemented by huge doses of brewer's yeast and regular injections of vitamin B_1 and liver concentrates. Twenty of his 54 patients, treated in this way, showed signs of "clinical recovery".

Stimulated by these attempts, Snell (1940) modified the Patek regime in a way which is extremely suitable to our local conditions, where various forms of cirrhotic liver confront us daily with therapeutic problems. His diet contains 500 gm. carbohydrate, about 60 gm. fat and 110 gm. protein, derived from other sources than meat, providing about 3,000 calories per day. The protein supply was covered by milk, egg-white, vegetables and wheat, supplemented by crude liver extract and yeast concentrates. Snell's results seem to be about equally as promising as those achieved by Patek.

We are at present investigating the influence of rest, anti-malarial treatment and yeast on the impaired *liver function* in *chronic malaria.* We would like to improve the function of these damaged livers by administration of amino-acid concentrates (Elman and Weiner, 1939) such as amigen and parenamine, as soon as they become available, being convinced that the majority of our cirrhosis cases develop under the combined impact of malaria, perhaps dysentery, and protein deficiency. The excellent effect of parenteral nutrition with such protein hydrolysates on the victims of the last Bengal famine are well known (Narayanan and Krishnan, 1944, Krishnan, Narayanan and Sankaran, 1944).

Many other therapeutic advances cannot be even mentioned here. All the new applications of whole blood, plasma and plasma concentrates, fibrin and gamma globulin will be reviewed as soon as we had an opportunity to use them.

Thiouracil

But one of the new drugs should find passing mention, at least, because of its great importance in reclaiming a group of pathological conditions for the physician. Thiouracil, a combine of a sulphonamide with thiourea, cures hyperthyroidism, exophthalmic goitre and toxic adenoma, in a high percentage of cases. The large doses, formerly applied, frequently caused leukopenia and even agranulocytosis (seven fatalities in 2,000 cases) which perhaps could be prevented by simultaneous liver administration or pyridoxine (Cantor and Scott, *loc. cit.*). The present routine dosage, however, which consists of 0·6 gm. per day in divided doses (Newman, 1945) or even 0·2 gm. only

(Thomson and Malins, 1945) until the basal metabolism is controlled, and 0·1 to maximum 0·4 gm. per day as a maintenance dose (Newman, *loc. cit.*) seem to be fairly safe.

Anti-Malarial Drugs

Concluding this incomplete review with a few remarks on malaria, public enemy number one of this country, we have to mention first the brilliant work of Hamilton Fairley. His investigations, carried out during the war at Cairns, Australia, on Australian volunteers, show that atebrin is the anti-malarial drug of choice, as long as paludrine is not available. In suppressive doses of 1 tablet (0·1 gm. or gr. 1½) per day, taken for six days every week for one week before and up to 23 days after exposure, atebrin not only prevents but permanently cures malignant tertian malaria in cent per cent of cases, so that neither fever nor, of course, black water fever occurred in any of those so treated. The same dose of atebrin completely suppresses the outbreak of benign tertian malaria as long as it is used, while a few weeks after stopping its administration overt attacks develop which require treatment with full doses. These doses, as recommended by a special committee of the Council on Pharmacy and Chemistry of the American Medical Association, are 2 tablets (0·2 gm. or 3 gr.) 5 times in 6 hourly intervals, *i.e.*, 10 tablets in 24 hours, followed by 1 tablet 3 times a day for 6 days, the whole course lasting 7 days and consisting of 28 tablets. To avoid recurrence of fever attacks it is rational to follow up such a course after two days interval with plasmoquine or pamaquine, 1 tablet of 0·01 gm. (gr. 1/6) twice a day for three to four days, while the patient keeps strict rest in bed to avoid circulatory accidents. Plasmoquine medication is required not only to destroy the sexual cycle of the plasmodium and so to prevent further mosquito infection but, especially, to clean the reticulo-endothelial system of the exo-erythrocytic cryptozoites, those forms of *Plasmodium vivax* which are responsible for relapses of benign tertian malaria. There is an important difference between falciparum cryptozoites which are readily destroyed by atebrin and those of the vivax species which are susceptible only to plasmoquine.

Hamilton Fairley's experimental work included also investigations of the suppressive value of quinine. He found that doses of gr. 5 per day had no such effect whatever. Daily doses of gr. 10 had neither supressive nor, of course, curative effect in falciparum infections but suppressed benign tertian attacks in two-third of his volunteers as long as this dose was daily administered; shortly after cessation unmitigated malaria attacks occurred in all these cases.

The tremendous effect of regular atebrin prophylaxis, carried out under military discipline, is illustrated by Hamilton Fairley's statement that in the Australian forces in New Guinea the malaria rate per annum was 740 per 1,000 in December, 1943, against 26 per 1,000 in November, 1944; that is roughly thirty to one.

Still more spectacular results in preventing malaria, are due to the discovery of the insecticidal action of *D D T*, which stands for dichloro-diphenyl -tricloroethane. This substance was synthesized by a German chemist in Strassburg as far back as 1874; for almost 70 years it was not used at all. In 1940 chemists of the J. R. Geigy company (Switzerland), searching for a new moth killer, found that it readily destroyed flies. An American military attachee secured a quantity of it for the U.S.A. where the entomologists of the Orlando Laboratory (Florida) discovered all we know about it. Sprayed as a 5 per cent solution in kerosene oil, it reduces mosquitoes and flies to zero or, at the maximum, 2 per cent of their previous number for at least one month; when used in larger quantities, this effect can be easily extended to 2 to 3 months. Added to larvicide oils, it multiplies their effect against anopheles larvae dramatically but not for a long time. It is by far the strongest remedy against cockroaches and bed bugs and capable of giving full protection against them. It offers the solution of the terrific problem of epidemic typhus by destroying with deadly precision all the lice when dusted under clothing in a concentration of 10 per cent of any mineral dusting powder or when used for impregnating underwear from which it can not be removed by soap and water for eight launderings. Recently I.C.I. claims to have synthesized a still more powerful insecticide, marketed as Gammexane. The paramount importance of these new drugs cannot be overrated. They are apt to change the living conditions in tropical countries even more than air conditioning promises to do.

Paludrine, the new anti-malarial drug of I.C.I., which was tested by the same research team under Hamilton Fairley, is said to be 10 times more effective than quinine, and to have thrice the effect of mepacrine; it is not clear what these comparative figures exactly mean as nobody knows how much quinine or mepacrine is required to cure a malarial infection. The facts, as given in a statement by Brig. N. Hamilton Fairley (19-12-45), are that it is a true causal prophylactic against falciparum and a powerful supressant of vivax malaria. Three tablets (0·1 gm. or 1½ gr. each) taken 3 hours before a volunteer was bitten by 10 mosquitoes, heavily infected with *P. falciparum*, prevented the outbreak of malaria and definitely cured every single case, provided one tablet was taken every day as long as exposure lasted; for how many days after the last exposure is nowhere stated. Plasmodia are completely destroyed before they enter the circulation as neither blood films nor blood transfusion to healthy volunteers ever revealed the presence of falciparum plasmodia in those treated with paludrine. This is a definite advance over atebrin, as inspite of atebrin suppressive treatment a few plasmodia regularly appear in the circulation between the 7th and 10th day after infection, although they are not numerous enough to lead to clinical manifestations. The curative effect of paludrine in malignant tertian malaria is excellent. One tablet cuts short a fever attack and 64 out of 65 cases were permanently cured by a course of 30 tablets (1 tablet thrice a day for 10 days).

Less satisfactory results are achieved in benign tertian malaria. The suppressive effect is complete as

long as 1 tablet is taken every day. But out of 31 cases, who received 10 tablets daily for 14 days, five relapsed; it is noteworthy that even such large doses did not have any toxic by-effect.

Summary

Summarizing, we see that sulpha drugs and penicillin have reduced the mortality rate of pneumonia, meningitis, strepto- and staphylococcal septicaemia, cholera and dysenteries to a small fraction of the previous amount.

The synthetic anti-malarial drugs and D D T are capable of preventing a high percentage of the malaria deaths, numbering at present some 30,00,000 per year. D D T will substantially lower the incidence of louse typhus and relapsing fever, of trypanosomiasis, filariasis and yellow fever; it will go a long way in controlling fly-borne diseases such as typhoid, amoebiasis, bacillary dysentery and probably, poliomyelitis. It could, intelligently applied, eliminate rat fleas so completely that plague would cease to constitute a public health problem. In other words, D D T could prevent every year 5,000,000 deaths and 400,000,000 cases of serious diseases.

It may be added that in the U.S.A. the death rate of tuberculosis in 1940 was roughly one-fifth of its rate in 1910, and the mortality rate of diphtheria in 1943 less than 1: 100,000.

Such figures induce not only pride in the ingenuity of man but also grave concern about the way, this immortalized species of ours will use its fast increasing numbers. It is true that on the other hand the mortality due to cancer has doubled and that of cardiovascular diseases trebled in the last 30 years and that birth control may offset partly the possible multiplication of the world population. And yet, with the elimination of almost all contagious diseases, the population pressure will increase to such an extent that only true humanization of world relationship could lead into peaceful channels the tremendous energies which owe their origin to medical progress.

References

Aykroyd, W. R.—*Indian M. Gaz.*, **80**:68, 1945.
Banting, F. G. and Best, C. H.—*J. Lab. & Clin. Med.*, **7**:251, 1922.
Bunn, P. A., Mc Dermott, W., Hadley, S. J. and Carter, A. C.—*J. A. M. A.*, **129**:320, 1945.
Butenandt, A.—*Naturwiss*, **17**:878, 1929.
Cantor, M. M. and Scott, J. W.—*J. Canad. M. A.*, **52**:368, 1945.
Carter, T. J.—*J.A.M.A.*, **127**:96, 1945.
Chain, E., Florey, H. W., Gardner, A. D., Heatley, N. G., Jennings, M. A., Orr-Ewing, J. and Sanders, A. G.—*Lancet*, **2**:226, 1940.
Doisy, E. A., Veler, C. D. and Thayer, S. A.—*Am. J. Physiol.*, **90**:329, 1929.
Domagk, E.—*Deutsche med. Wchnschr.*, **61**:250, 1935.
Elman, R. and Weiner—*J.A.M.A.*, **112**:796, 1939.
Feldman, W. H., Hinshaw, H. C. and Mann, F. C.—*Am. Rev. Tuberc.*, **52**:269, 1945.
Finland, M., Meads, M. and Ory, E. M.—*J.A.M.A.*, **129**: 315, 1945.
Fleming, A.—*Brit. J. Exper. Path.*, **10**:226, 1929.
Hamilton-Fairley, N.—*Tr. Roy Soc. Trop. Med. & Hyg.*, **38**:311, 1945.
Hargreaves, W. H.—*Lancet*, **2**:68, 1945.
Heilig, R.—*Indian M. Gaz.*, **78**:129, 1943.
Helmholz, H. F.—*Proc. Staff Meet. Mayo Clin.*, **20**:357, 1945.
Johnson, B. A., Anker, H. and Meleney, F. L.—*Science*, **102**:376, 1945.
Kabat, H.—*Pub. Health Rep.*, **59**:1635, 1944.
Keefer, Ch. S.,—*Am. J. Med. Sci.*, **210**:147, 1945.
Kennedy, F. and Wolf, A.—*Arch. Neurol & Psychiat.*. **37**:68, 1937.
Idem—J.A.M.A., **110**:198, 1938.
Krishnan, K. V., Narayanan, E. K. and Sankaran, G.—*Indian M. Gaz.*, **79**:160, 1944.
Minot, G. R. and Murphy, W. P.—*J.A.M.A.*, **87**:470, 1926.
Narayanan, E. K. and Krishnan, K. V.—*Indian M. Gaz.*, **79**:158, 1944.
Newman, E. V.—*Med. Clin. North America*, **29**:302, 1945.
Patek, A. J.—*Proc. Soc. Exper. Biol. & Med.*, **37**:329, 1937.
Idem—Bull. N. Y. Acad. Med., **19**:498, 1943.
Ross, S. and McLendon, P. A.—*J.A.M.A.*, **129**:327, 1945.
Saxl, P. and Heilig, R.—*Wien. klin. Wchnschr.*, **33**:943, 1920.
Schatz. A., Bugie, E. and Waksman, S. A.—*Proc. Soc. Exper. Biol. & Med.*, **55**:66, 1944.
Shallenberger, P. L., Denny, E. R. and Pyle, H. D.—*J.A.M.A.*, **128**:706, 1945.
Siegel, M., Karr, H. V. and Julianella, L. A.—*Am. J. Hyg.*, **41**:228, 1945.
Smith, M. L. and McClosky, W. T.—*Pub. Health Rep.*, **60**: 1129, 1945.
Snell, A. M.—*Minnesota. Med.*, **23**:551, 1940.
Spies, T. D.—*J. A. M. A.*, **111**:584, 1938.
Thomson, A. P. and Malins, J.—*Practitioner*, **155**:193, 1945.
Walker, M. B.—*Lancet*, **1**:1200, 1934.

XXIII ALL-INDIA MEDICAL CONFERENCE, MADURA

The 23rd All-India Medical Conference will be held at Madura, Madras Presidency, during the Christmas Week (December 1945) under the auspices of the Indian Medical Association.

In addition to Business Meetings and Scientific Sessions, an All-India Medical Exhibition will be held for a week. Sight seeing in and around Madura will be arranged. Conducted tours or trips to Rameswaram and Trivandrum will also be arranged. A special train from Madras to Madura and back can be arranged. Programme of tours and charges will be announced later.

M. V. Natesan
A. K. Rajagopalan
P. A. S. Raghavan (Transport)
General Secretaries.

P. N. Ramasubramanyam,
Chairman, Reception Committee.

JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION
CALCUTTA, JUNE, 1946

DENTAL EDUCATION AND THE BHORE COMMITTEE REPORT

In modern medical practice the oral hygienic condition is seen to as a routine measure and there is no difference of opinion that if we are to get the optimum results in treatment the co-operation of the dental surgeon is essential. Although medical education was started in the year 1858, it was not till the year 1920, that the first dental college in India, the Calcutta Dental College and Hospital was established, by private initiative and effort. It is worth noting that after two hundred years of enlightened administration no efforts at imparting dental education was made till the people awakened to the needs of the times and tried in their humble way to help themselves. The Bhore Committee draws attention to the above fact and mildly comments[1], "Dentistry has unfortunately been one of the neglected subjects of study in practically all Indian Universities".

It is a pleasure to find that the Bhore Committee has gone into the whole problem of dental education and dental service to the masses and has made definite recommendations to improve the present conditions. The problem may be gauged from the fact that at the present moment there is one qualified dentist to every 300,000 of the population. At the present time there are only four dental colleges in India, *viz.*, Calcutta Dental College & Hospital, Nair Hospital Dental College, Bombay, Currimbhoy Ebrahim Dental College, Bombay and de Montmorency Dental College, Lahore, and the average outturn of graduates from these is not over 60-70 annually.[2] If three more dental colleges are established in Lucknow, Patna and Madras during the next five year period and if all these seven each train and pass out 100 qualified dentists every year it will take over 70 to 75 years to reach a proportion of 1 qualified dentist to every 5000 of the population. The vastness of the problem can be visualized from the above facts. So the Bhore Committee has recommended that in the first five year period, the present Colleges in Calcutta, Bombay and Lahore should be taken over by the State (the Lahore Dental College being a Government institution), new buildings built and new equipment bought to make these three up-to-date modern dental training centres, each capable of teaching 100 students in each year or 400 students in all. The Bhore Committee rightly points out that due to the paucity of trained teachers in India facilities should be provided for selected number of dental surgeons from this country to undergo advanced training abroad.[3] The initial capital cost of each dental college has been worked out at Rs. 25 lacs for land, building and equipment and the recurring expenses for each college at 5 lacs per year per college,[4] for training 100 dental surgeons and 100 dental hygienists every year. The only snag in this programme is that unless the provinces find the money to run these colleges, the whole scheme or schemes will remain in the air or in the archives of the secretariat. We, therefore, feel the necessity of drawing the attention of the provincial governments (because it is expected that in the new order of things, the provinces will be supreme in the Health field) to this portion of the recommendations of the Bhore Committee and trust they will implement the schemes, so carefully worked out.

The object of the expansion of dental education is not only to furnish personnel for private dental practice but also to provide personnel for Provincial Dental Services which will cater for that high proportion of the population, which is at present completely neglected. The short term dental programme will attempt to provide (1) dental sections at all secondary health unit hospitals that will be established during the short term and (2) a travelling dental organization for the area covered by each secondary unit.[5]

The Committee is distinctly of the opinion that unless the state itself undertakes the establishment of a comprehensive dental service to meet the urgent needs of the population, the increase in the number of dental colleges and the improvements in the training of dental surgeons and dental hygienists will not lead to satisfactory results.[6]

The Bhore Committee has hit the nail on the head when it has discussed Dental Legislation and opined that unless there is a comprehensive all-India dental legislation for compulsory registration of dentists and prohibition of practice by unregistered men, dentistry as a science can make little real progress.[7] Those of our citizens that go about our big cities with eyes open must have noticed the quacks and charlatans, foreigners and indigenous, who without any knowledge of asepsis and sterilization carry on the practice of dentistry, without any let or hindrance.

Even small British colonies, like Ceylon, West Indies, or Kenya have dental laws and no one may practise dentistry unless he is properly qualified and registered. At present India is the happy hunting ground of quacks as well as of foreign refugees of all description, into whose countries, an Indian national may not enter, not to speak of practising dentistry. The only province in India where there is any Dental Act is Bengal but the Bengal Dentists Act is defective in that it does not compel registration nor is there any bar to practise by unregistered men. The Bhore Committee has seen through this and has rightly advocated that in any progress in the dental field in India, the first pre-requisite is dental legislation leading towards compulsory registration and prohibition of practice by unregistered men. This is the crux of the whole problem, unless this is done, no progress in dental education is possible.

[1] Report of the Health Survey and Development Committee, 1946, Vol. I, p. 166.
[2] *Ibid.*—Vol. I, p. 167.
[3] *Ibid.*—Vol. I, p. 168.
[4] *Ibid.*—Vol. III, pp. 248 & 328.
[5] *Ibid.*—Vol. II, p. 380.
[6] *Ibid.*—Vol. II, p. 380.
[7] *Ibid.*—Vol. II, p. 379.

CURRENT MEDICAL LITERATURE

Some Causes of Syncope

Fainting or syncope is a common enough occurrence, but it has many possible causes. A study of these is the basis of a recent article by Engel, Ferris, and Romano (*Cincinn. J. Med.*, **26**:93, 1945). Some causes are rare, but their possibility must be borne in mind. The ultimate reason for the loss of consciousness is cerebral anæmia. The two contributory causes of this are fall in blood pressure and diminished output of blood from the heart. Failure of return of blood to the heart, due to pooling, loss of vasomotor tone, fall in rate, contributes to these defects. In the majority of cases the diagnosis can be made with a fair degree of certainty by clinical means alone if the patient is seen at the time; a careful history of the circumstances of the attack and of any premonitory symptoms may even provide a diagnosis in retrospect. The commonest group is that in which fainting results from a fall in blood pressure, which the authors call the vasodepressor type. This includes what is more commonly known as the vaso-vagal faint. The relative importance of the vagal factor varies. In very sudden faints it may predominate, as Lewis pointed out long ago. In others the onset is more gradual and the vasomotor side is dominant; such cases may be purely psychological in origin. Sometimes a depressor reflex from the carotid sinus is over-active. This carotid sinus syndrome has attracted much attention, but in all probability it is rare, although a sensitive carotid sinus is not uncommon in the elderly. An interesting, though rare, type is true orthostatic hypotension; here there seems to be no control over the distribution of the blood in relation to posture. Possibly some lesion of the central nervous system lies behind it, or of the sympathetic. It can be caused artificially by very extensive sympathectomy. Complete cardiac standstill provides another cause. This may be reflex and provoked by stimulation of the carotid sinus. The standstill may affect only the ventricles, the auricles continuing to beat. These cases provide the Stokes-Adams' syndrome. Mnay are due to failure in auriculoventricular conduction; but records rarely obtainable, have shown that in others ventricular fibrillation or tachycardia is present. In a unique case described by Bruce Pearson (*Brit. Heart J.*, **7**:85, 1945) there were prolonged complete cardiac standstill and profound bradycardia quite unaffected by atropine or anything else. Possibly, in this instance, the sinus was in a state of diminished excitability.

Occasionally fainting results from over-ventilation of the lungs. These patients are closely linked with those in whom hysterical fainting occurs. There is no change in the pulse rate, in colour, or in blood pressure; nor does the encephalogram show the change to high voltage deflections which are constantly recorded in true syncope, thus differentiating hysterical "fainting" from any real circulatory disturbance. In old people syncope may be prolonged and recovery slow, perhaps because of cerebral arteriosclerosis. The only valvular lesion with which syncope is at all common is aortic stenosis, and that the type occurring in the elderly fibro-calcareous stenosis.

The authors stress the following points in the history: frequency of faints, posture, nature of provocative stimulus, duration, focal neurological symptoms, rate of recovery, biting of tongue, or incontincnce of sphincters; changes in colour, convulsive movements, sweating, changes in pulse and blood pressure; and physical examination of the heart. Information on these points will usually provide a diagnosis. This is important, for fainting is always an alarming event for laymen and tends to arouse serious doubts as to the state of the heart. It will be seen that most cases are not serious in nature, but that a small minority are—(*Brit. M. J.*, **2**:772, 1945).

Insomnia Due to Left Ventricular Heart Failure

Wheeler and White (*J.A.M.A.*, **129**:1154, 1945) write that insomnia as a presenting symptom of pulmonary vascular congestion has been generally neglected, because obvious dyspnœa, orthopnœa or Cheyne-Stokes' respiration is usually also present and dominates the clinical picture. Insomnia may, however, be an early and predominant symptom of pulmonary congestion due to left ventricular failure in cases in which dyspnœa is not a prominent complaint. In these cases the insomnia is likely to be misjudged as a symptom of heart failure and ascribed to some other cause. Especially is this true in the absence of basal rales. The insistence of the patient on sitting up, which is evidence of ill defined orthopnœa, in the presence of physical signs of left ventricular weakness (gallop rhythm, pulsus alternans and accentuation of the pulmonary second sound) may lead to a recognition of the etiology of the sleeplessness.

Thus it behoves the physician dealing with patients who show features of strain on the left ventricle, namely hypertension in particular, aortic valve disease (stenosis or regurgitation) and extensive myocardial infarction, acute or chronic, to be constantly on guard to watch for early dyspnœa, either on effort or as a cause of insomnia, so that adequate treatment may be speedily prescribed to combat the left ventricular weakness and failure which are responsible. The significance of obvious dyspnœa is common knowledge and receives as a rule adequate treatment, but the insomnia is likely to be misinterpreted and vainly or inadequately controlled by hypnotics, often causing a serious delay of days or weeks before the signs of increasing heart failure become so evident that radical or emergency treatment proves necessary. At such later stages of heart failure insufficiency of the right ventricle (as shown by increased systemic venous pressure, liver enlargement and dependent œdema) tends to appear, and if such right ventricular failure becomes pronounced the pulmonary congestion, and with it the insomnia, may lessen, but painful liver enlargement may take its place and in its turn also cause some insomnia. Rest and digitalis alone may suffice to control the orthopnœa and insomnia of early left ventricular failure, but sometimes it is necessary to resort to diuretics, especially the mercurials, in repeated dosage and to a very low salt diet.

Paroxysmal Abdominal Pain: A Form of Focal Symptomatic Epilepsy

Moore (*J.A.M.A.*, **129**:1233, 1945) writes that clinical study of 6 patients (2 children, 4 adults) in whom paroxysmal abdominal pain was either the only symptom or the dominant one revealed this to be a type of epileptic variant (abodminal epilepsy), as shown by the pattern of symptomatology, electroencephalography and response to anticonvulsant drugs. Three of the patients presented the history of antecedent head injury; 1 had cerebral angoineurotic œdema following injection of antiserum prior to the onset of abdominal seizures; 1 had paroxysmal abdominal pain diagnosed as intestinal tetany occurring in an unrecognized case of tuberous sclerosis; 1 showed no eitologic background.

There is need for recognition of unexplained paroxysmal abdominal pain, when study of the abdominal viscera has been negative as being possibly of cerebral origin due to abnormal

cerebral discharges arising in the premotor or parietal areas. These symptoms are liable to be present in the returning war veteran who has suffered a severe head injury, and there is likelihood of failure to evaluate its true nature.

The criteria for diagnosis are based on (1) exclusion of intrinsic visceral disease, (2) adequate historical data, (3) attack pattern of an epileptic order, (4) associated epileptic show, (5) objective evidence of cerebral organic disease or dysfunction and (6) effect of anticonvulsant drugs on the symptom of abdominal pain and on the electroencephalogram.

Comparatave Effect of Sulphonamide and Penicillin in Pneumonia

Anderson and Ferguson (*Lancet*, **2**:805, 1945) write that a series of 126 patients over the age of 35 years suffering from pneumonia was divided by random selection into two groups, one of which was given sulphathiazole, the other penicillin. All patients received the same symptomatic treatment and general clinical control.

The distribution of factors known to influence the course of the disease was the same in the two groups.

Each method achieved fairly similar results. In 3 patients, however, it was thought that penicillin had produced an unexpected recovery.

There is no reason for abandoning properly controlled sulphonamide treatment for the average case; and it is suggested that penicillin should be used for selected patients, usually in combination with a sulphonamide. Indications for the use of penicillin are tentatively suggested. The cause of delayed resolution is discussed.

Some tentative guides for the efficient use of penicillin may be formulated. In the first place, in severe cases, an adequate dose of a sulphonamide should be given during the first twenty-four hours. The authors believe that results with sulpathiazole, are satisfactory for unselected patients in this age group, and that pneumonia in the person over 40 years should be regarded as a medical emergency. In the second place, a preliminary blood-culture (before administering sulphonamides) becomes of increasing importance; for a positive result, especially if the infection is heavy, should suggest the need for starting penicillin. Thirdly, a routine white-cell count, when treatment is begun, will be of value in picking up the leucopenic patient (not uncommon in this age-group) who may respond unsatisfactorily to sulphonamides. Lastly, but not least important, the clinical assessment of the patient should be taken into account. They are satisfied that a poor cardiovascular system remains the most important prognostic factor in pneumonia, and should constitute an indication for combined therapy. Extensive pulmonary involvement, severe cyanosis, and dyspnœa are other indications which may call for the support of chemotherapy by penicillin. The fact that we now have two effective methods of attack makes it more than ever important that the closest cooperation should be established between the laboratory and the clinician if the best results are to be obtained in the management of pneumonia.

Penicillin in the Treatment of Subacute Bacterial Endocarditis

Flippin and others (*J.A.M.A.*, **129**:841, 1945) give in the following lines the summary of their observations:—

Twenty cases of subacute bacterial endocarditis were treated over one year with penicillin. Since there have recently appeared several excellent papers in which the salient features of this disease have been discused, we will limit ourselves to several factors concerned with the management of these cases.

Results of Penicillin Therapy—Of the 20 patients with subacute bacterial endocarditis, 16 had definite subsidence of their infection, 12 are still alive and with 1 exception are all well. Of the 8 who did not survive 4 were completely free of active infection and died as a result of heart disease. Three patients in this group failed to respond to therapy, and the eighth died soon after treatment was completed as a result of an acute pulmonary embolus.

Establishment of Diagnosis by Positive Blood Cultures Before Starting Penicillin—Not infrequently a patient suffering with this disease is started on relatively small doses of penicillin before a positive blood culture is obtained. The dangers from this practice are threefold: 1. The organism may lose its sensitivity to penicillin. 2. The chances of obtaining a positive blood culture are lessened, despite the addition of penicillinase to the culture material. 3. The cardiac lesion itself undoubtedly progresses. Therefore it would seen best to withhold penicillin until a positive culture is obtained or, if the clinical diagnosis warrants, to administer adequate amounts of penicillin for the disease. In several cases not included in this series, in which routine blood cultures were negative, we obtained positive results by employing anaerobic and arterial cultures.

Adequate Penicillin Dosage—At this writing we are unable to make any conclusive statement regarding the amount or route of administration of penicillin for the treatment of this disease. As indicated, we employed 300,000 units a day for fourteen days as the initial course. This appeared adequate in 14 of the 20 cases. However, in view of subsequent experience with 16 additional cases of the disease, not included in this report, we believe that all patients with subacute bacterial endocarditis should receive at least 500,000 units a day for at least five weeks.

We have used a continuous intravenous infusion of penicillin where possible, but we have no reason to believe that this method is any better than the intermittent intramuscular route. Sternal infusion of penicillin is practical for limited periods of time.

Eradication of Possible Foci of Infection—As a precaution against reinfection, a careful search for possible foci of infection, such as infected teeth and tonsils, should be made, and, if surgical procedure is warranted, the patients are given full doses of sulphonamides or, preferably, penicillin before and after operation.

Penicillin X—In a case in which the infecting organism had become highly resistant to penicillin, we employed penicillin X with apparent success.

Effect of Glutamic Acid on Petit mal Types of Epilepsy

The observation that grand mal attacks of epilepsy are in the majority of the cases more or less completely controlled by anti-convulsant therapy, whereas petit mal types of attack not infrequently are not influenced by medication, suggests that the trigger mechanism in the two types may not be identical. The petit mal seizures respond favourably to fasting and to a ketogenic diet. The difficulties however, in maintaining a dietary regimen that will produce ketonuria led to a search for acidosis-producing drugs. From theroretical considerations, Waelsch thought that *dl*-glutamic acid or *l*-glutamic acid

should be capable of producing acidosis, Furthermore, *l*-glutamic acid is known to be metabolized by slices of brain tissue. Price and his associates (*J.A.M.A.*, **122**:1153, 1943) found that 4 Gm. of *dl*-glutamic acid administered three times daily was sufficient to maintain the pH of the urine at about 5·0. Administration of this amino acid together with the usual anticonvulsant therapy (diphenyl-hydantoin-phenobarbital combination) was effective in decreasing the frequency of petit mal seizures in their patients. There was also a pronounced mental and physical alertness. Grand mal attacks were not affected by the drug. Spangler (*Ann. Allergy*, **3**:241, 1945) administered glutamic acid to 6 patients subject to both grand mal and petit mal seizures and selected because their petit mal attacks were not influenced by the anticonvulsant drug therapy. When glutamic acid was added to the crotalin-diphenyl-hydantoin-phenobarbital therapy the petit mal seizures were reduced in frequency and modified in character and severity. The effect was practically the same in all 6 patients. The amount of acid administered was guided by a daily recording of the urinary pH in an effort to keep the hydrogen ion concentration below 5·0. The pH of one of these patients was recorded daily for one year. The beneficial effect of glutamic acid in lowering the pH of the urine in epilepsy, as illustrated in this case, and the fact that menstruation, even when the patient is taking glutamic acid, often causes an increase in the petit mal seizures, suggests that endocrine function may be less of a factor in the eitology of petit mal than the increase in the hydrogen ion concentration of the urine during menstruction. It is possible, Spangler believes, that petit mal seizures may result more from an altered immunologic allergic response, as shown by a decreased hydrogen ion concentration in the urine.—*J.A.M.A.*, **129**:1021, 1945.

Streptomycin and Promin in Experimental Tuberculosis

Guineapigs infected with tuberculosis were employed by Smith and McClosky (*Public Health Rep.*, **60**:1129, 1945) to determine whether streptomycin has any beneficial effect, to compare the results of streptomycin therapy with the sulfone, promin and to explore the possibilities of combined treatment with these two chemotherapeutic agents. Under the experimental conditions of treatment streptomycin injected intramuscularly produced a definitely greater chemotherapeutic effect than promin. The dose of promin used was about one-half the maximum tolerated dose, while the dose of streptomycin used was less than one twentieth of its maximum tolerated dose; hence streptomycin had a therapeutic index at least ten times that of promin. Neither of these agents used alone completely eradicated the tuberculous process. Larger doses and better methods of administration may well increase the therapeutic effect of streptomycin. Smith and McClosky obtained results better than any they had ever achieved before in the treatment of experimental tuberculous infections. They believe these experimental results warrant the cautious application of combined treatment to selected patients.—*J.A.M.A.*, **129**:620, 1945.

Phthalylsulphathiazole in Infectious Diseases of the Colon

Streicher (*J.A.M.A.*, **129**:1081, 1945) writes that phthalylsulphathiazole is less toxic and more bacteriostatic than any intestinal agent used previously and that, because it has these properties, smaller doses of this drug may be used to advantage. In their analysis, determinations have been made to show that a daily dose of 3 Gm. taken orally will bring about the desired therapeutic effect in infectious diseases of the colon and that doses of 12 Gm. or more are not essential. Large amounts of phthalylsulphathiazole not only are wasteful but are detrimental.

Patients receiving 12 Gm. of the drug complain of considerable cramping in the abdomen, exaggeration of the liquid consistency of the stool and an increase in the number of evacuations. In a comparative study made of phthalylsulphathiazole and succinylsulphathiazole one comes to the conclusion that the new sulphonamide is a superior therapeutic agent in colon infections.

Despatch of Material for Histological Examination

Hay (*Edinburgh M.J.*, **52**:426, 1945) gives the following instructions for despatch of the material:—

Take over the material[1] from the operator.

Wrap it up temporarily in sterile cloth soaked in 10 per cent formalin fixative[2] or, if small, pass straight into a specimen tube[3] filled[4] with formalin fixative and apply screw top or cork.

Examine the large specimen gently and, if necessary, make one or more parallel transections in its long axis, the first plane of which should pass through lesion, skin or mucosa, nipple, hilum of organ, anatomical guide post, surrounding structure, etc., so as to give a clear view of anatomical relations.

Reconstitute the specimen for despatch to the pathologist, wrapping it in 10 per cent. formalin soaked cloth or gauze, covering it with waterproof jaconet or cellophane and enclose the whole with its packing in a tin or box.

If the specimen cannot be sent as a whole, remove one or more suitable portions to include advancing margin or capsule, diagnostic centre, unaffected tissue and adjacent structures useful for localisation and diagnosis, such as skin, bone, mucosa, extensions to lymph nodes, metastases, etc.

Drop the selected portions, of not less than one quarter inch thickness[5] into specimen tube containing formalin fixation.

Fasten the cork or screw top securely with adhesive tape to prevent leakage in the post.

Pack the specimens along with sufficiently full and legible[6] notes which should include, at least, age, sex, married state, time of removal and tentative diagnosis; but these notes are greatly improved by further relative clinical detail.

Despatch as soon as possible[7] to the pathological laboratory.

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[1] It is better not to wash the specimen unless much covered with blood. If the material has been deposited on dry gauze, get it detached as quickly as possible and into fixative to prevent capillary-drainage desiccation by the gauze. Drying ruins the specimen especially when small.

[2] Composition: formalin 1: tap water 9.

[3] If the specimens are fragmentary it is advisable to tie them all up into a little bag of linen (not gauze) and place the whole thus protected from dispersal, in the fixative.

[4] Inclusion of any appreciable volume of air should be avoided, because it is apt with the fluid to cause injury to the material in transit.

[5] If the specimen is too thin it is difficult to embed it and is apt to curl up.

[6] Block letters for the name of patient and for signature are advisable. The name of the chief is more important for laboratory record than that of the resident.

[7] It may be that, for reasons of economy, several pieces of material are retained in fixative for some days so as to be despatched in one parcel. Retention in formalin for too long causes undesirable shrinkage of the tissue.

Expect a report in five or six days[8] after receipt unless prolonged treatment such as decalcification is necessary.

Add any special information required.

Regional Segmented Colitis

Barbasa and others (*Pro. Staff Meet Mayo Clinic*, **20**: 134. 1945 Ref. *Am. J. Digest. Dis.*, **12**: 351, 1945) write that the term "chronic ulcerative colitis" refers to a group of different disease entities among which group is included "segmental" or "regional" colitis. Regional colitis refers to a "nonspecific or cryptogenic inflammatory ulcerative or hyperplastic lesion involving one or more short or long segments of large bowel". The terminal portion of the bowel is spared. The lesion may be continuous or consist of multipile involvements. The ileum is either not involved at all or is involved secondary to the main colonic lesions. At the Mayo Clinic segmental colitis constituted four per cent. of all cases of ulcerative colitis. The oldest patient was 73 years, the youngest three years and the sex distribution was equal.

The origin of the regional colitis still remains unknown. All experiments on the subject have yielded either negative or dubious results.

The authors discuss the characteristic lesions they found in their series of patients coming to surgery and present an outline of the clinical course of the patient. They advocate medical treatment of some sort for most patients. Rest, proper diet, supportive measures including blood transfusions are recommended. If surgery is advisable, then radical operation is the best treatment. The authors emphasize that it is essential to resect the bowel well beyond the apparent limitation of the disease. Although roentgenographic studies appear to show a sharp demarcation line, and direct examination at operation may likewise reveal a sharp limitation area, careful inspection will reveal extension of the disease processes for 4 to 6 inches beyond. Stippling of the serosa without thickening of the bowel, due to minute puncture hæmorrhages, is found distal to the involved area. The stippled segment must also be removed or recurrence will ensue. The reason for the spread of the regional colitis disturbance is not known: It appears to be due to direct extention and not to spread by the lymph vessels.

Renal Calculus

Gray (*Med. Press & Cir.*, July 1945, *Med. World*, **63**:626, 1945) says that in order to prevent the formation of stones the fluid intake should be increased and the food intake reduced to as low a level as possible. Uric acid stones are more likely to be retained in solution in alkaline urine. We should aim, therefore, in such cases to give a reduced protein and a large vegetarian diet. Alkaline drinks and diuretics are also helpful. When the urine contains phosphates in excess the urine should be acidified by giving a relatively high protein diet and an acid sodium phosphate mixture of ammonium chloride. In the case of oxalates the urine should be alkalinised and any tendency to dyspepsia and intestinal fermentation corrected.

[8] Frozen sections are quick but may not be reliable. A stained, thin, paraffin section of already fixed tissue can be put through for report in twenty hours, but should only be asked for in case of special urgency, since it requires individual attention throughout.

Treatment of Gonococcal Arthritis with Sulphonamides and Artificially Induced Fever

Solomon (*Am. J. Syph. Gon. & Ven. Dis.*, **29**:567, 1945) reports that twenty-three cases of drug resistant gonococcal arthritis are reported. Of these, ten patients were treated with sulphonamides and artificial fever in the hypertherm with the following results: four were cured and the remainder showed either considerable or partial improvement. Fifteen were treated with sulphonamides and artificially induced fever with intravenous typhoid vaccine with the following results: six were cured, six were either considerably or partially improved; three were not benefited; two of the latter were subsequently treated in the hypertherm with good results.

The result with both of these methods was encouraging. The use of the hypertherm is preferable to the use of vaccine. The most important consideration, however, is early treatment. Hence, if the hypertherm is not available, combined vaccine and sulphonamide therapy should be instituted without delay.

It is emphasized that specific therapy does not supplant the regular methods of management of arthritis which include bed rest, special diet and physiotherapy. Specific therapy is used in addition to these and results in a much greater percentage of cures and in the restoration of joints to useful function.

Effects of Roentgen Ray on the Human Brain

Wachowski and Chenault (*Radiology*, **45**:227, 1945) write that six cases showing definite signs and symptoms of brain damage due to high voltage roentgen radiation have been presented. Three of the cases came to autopsy and showed extensive degenerative changes in the nerve tissue. One of the cases showed damage with a dose that is often quoted as being safe for brain tissue.

The striking damage to the nerve tissue did not have a counterpart in the vascular structures.

The doses used failed to destroy all of the tumor in any of the autopsied cases of this report, as well as in other unreported cases.

Roentgen Therapy of Primary Neoplasms of the Brain and Brain Stem

Price and Others (*Radiology*, **45**:247, 1945) write that definite clinical improvement is afforded by rœntgen therapy of malignant primary nœplasms of the brain and brain stem.

Twenty-five of 60 patients with complete follow-up over the past four years are either "alive and well" or "alive and well save for residual symptoms."

No deleterious effects chargeable to the rœntgen therapy have been observed in the series. Primary regrowth of hair has been excellent. No late degenerative changes have been observed following total tumor doses of 15,000 r.

Glioblastoma multiforme continues to show a high mortality rate despite increasingly large doses of rœntgen radiation. Probable improvement in longevity is indicated by the analysis of the data. Tumor doses in excess of 7,500 r. will be required.

The effect on *astrocytoma* is not so marked as would be expected but this should not discourage treatment similar to that recommended for glioblastoma.

Choice of Local Burn Therapy

The following table (*Am. J. Sug.*, p. 64, 1944, Ref. *Antiseptic*, **43**:69, 1946) gives the local treatment of burns.

Region	*First Degree*	*Second Degree*	*Second and Third Degree*
Face	Vaseline dressing Triple dye Sulfadiazine ointment	Vaseline dressing Pressure treatment (Koch) Saline packs Sulfadiazine emulsion	Vaseline and pressure treatment Saline packs
Hands	Boric acid; vaseline paste dressing Sulfonamide paste Dye therapy	Vaseline dressing Pressure treatment Saline baths	Vaseline and pressure dressing Saline baths
Extremities	Sulfonamide paste Triple dye Tanning methods	Pressure treatment Sulfonamide paste Tanning methods Saline baths	Pressure treatment
Trunk	Tannic acid-silver nitrate Sulfadiazine spray Triple dye Vaseline dressing	Tannic acid-silver nitrate Sulfadiazine spray Triple dye Pressure dressings	Tannic acid-silver nitrate Pressure dressings Saline baths
Perineum and Genitalia	Boric acid ointment Sulfonamide paste or emulsion Dye therapy.	Sulfonamide paste or emulsion Saline baths	Sulfonamide paste or emulsion Saline baths

Penicillin in Acute Osteomyelitis

Sir Howard and Lady Florey (*Lancet*, **1**:387, 1945) reporting their first therapeutic trial of penicillin in osteomyelitis, prophesied that a time might come when that disease, treated early and intensively with penicillin, would not require surgical intervention. Recent papers by Altemeier and Helmsworth (*Surg. Gynec. & Obset.*, **81**:138, 1945) and by McAdam (*Brit. J. Surg.*, **33**:167, 1945) suggest that the Floreys' early prophecy is close to fulfilment.

The more important conclusions reached in the two papers are in close agreement, and may be summarized briefly. (1) A suitable daily dose is 100,000 Oxford units administered by continuous intramuscular drip or by three-hourly injections. (2) Treatment should be continued for at least two weeks, or until three specimens of pus obtained by an intrametaphysial needle are sterile (McAdam). (3) Ancillary surgical measures should be limited to the immobilization of the affected limb, the incision of abscesses, the late removal of large sequestra, and the insertion on occasion of a sternal-puncture needle into the metaphysial focus. Marrow drainage, performed in five of McAdam's patients, and these not the most severely affected, was followed in at least two cases by wide sequestration and persistent sinuses. (4) Penicillin therapy should be established for twenty-four hours before any surgical procedure even the opening of an abscess, is undertaken. (5) With adequate penicillin administration an initially positive blood culture becomes negative after three days or so. (6) The patient's general condition improves, but only after a few days' treatment. (7) The initial temperature, continuing high for a few days, falls gradually to normal after a week or more. (8) Metastatic foci present when the patient is first seen respond well to suitable surgical procedures during penicillin administration; new metastatic foci are not likely to develop once treatment is begun.

The radiological record of the behaviour of an infected bone under the influence of penicillin is interesting. The characteristic feature is a mottled demineralization, which proceeds to a maximum translucency weeks, or months, after all infection has subsided, and which is ascribed to a post-infective absorption of lamellae killed during the infective period. Quite large sequestra sometimes fade gradually, and in both papers an analogy is drawn between this phenomenon and the absorption of sterile bonechips transplanted to fill a defect. In both circumstances demolition must be complete before reconstruction can proceed.

McAdam has inserted a stermal-puncture needle directly into the infected metaphysis, as suggested by Aird (*Proc. Roy. Soc. Med.*, **38**:509, 1945). Pus can be withdrawn immediately to decrease tension in the metaphysial abscess, and to permit precise diagnosis and an estimation of the penicillin sensitivity of the responsible organism. If the needle is left in place, and if specimens of pus are withdrawn at intervals, the time of sterilization, when penicillin administration can safely be discontinued, is precisely known. Further, the needle offers for continuous penicillin administration an alternative (intrametaphysial) route, by which can be attained not only a high blood level of penicillin but also a high local concentration, which may be particularly valuable if an adjacent joint—the hip, for example has been directly infected. Only one of McAdams 40 patients died, from an infection by a partially penicillin resistant *Staph. aureus*. Altemeier and Helmsworth, in 34 cases, had also one fatality—a case of severe hæmatogenous osteomyelitis of the tibia neglected for fourteen days; death took place seventeen hours after admission to hospital. The fatality rate of osteomyelitis is a notoriously fallacious criterion of the efficacy of a form of treatment, so much does the disease vary in severity from year to year and from lustrum to lustrum in an irregular epidemiological curve. Yet a fatality rate of 2·8% in the combined series of 74 cases, of which more than half had positive blood culture, supports McAdam's conclusion that "recovery can be anticipated in every patient with acute hæmatogenous osteomyelitis treated with penicillin, provided that the infecting organism is penicillin-sensitive, and the patient is not *in extremis* when first seen."—*Brit. M. J.*

Fibrositis of the Back

Copeman and Ackerman (*Quart. M. J.*, **13**:37, 1944) reported a clinical study of "fibrositis" of the back in which the pain was studied clinically and the occurrence of trigger points confirmed. The exact situation in which these occurred in a large number of patients was charted and the pain pattern of definite shape obtained. The dissection of 14 bodies of persons dying from various causes, excluding those of fibrositis, established the existence of a basic fat pattern. This fat pattern was observed to correspond exactly in shape and situation with the pain pattern already charted. Lesions suggestive of inflammatory reaction were not found in any deep fibrous tissue, but certain abnormalities affecting the lobules of the fat pattern were found in several instances. The abnormality which seemed most likely to have clinical significance was the herniation of certain fat lobules through weaknesses or actual deficiencies in the walls of their investing fibrous tissue. A series of 10 biopsies of selected patients suffering from fibrositis were reported in full. Although the authors thinks that the microscopic lesions described in the removed fat may be causative of the pain, they believe that its basic origin lies in an increase of tension in the fat lobules affected where they are invested by a nondistensible fibrous membrane. The anatomic lesions found were probably a later result of this condition where it persists. Further clinical support to the concept of herniation of fascial fat as a cause of back pain is offered by Herz (*J.A.M.A.*, **128**:921, 1945) who reports a striking relief of pain in 6 patients suffering from excruciating pain in the back. In all of his cases there was a history of trauma due to strain, which had initiated the pain. Pain in the back was extremely severe in all and in some instances was referred down one leg. In all cases a definite mass was palpable which was extremely tender, and pressure on it intensified the pain. Injection of these masses with procaine solution provided temporary relief in all instances. In these 6 cases, fairly wide dissection of fat in the painful area has relieved the pain whether or not the herniation could be clearly delineated. Mylechreest (*Ann. Rheumat. Dis.*, **4**:77, 1945) who dissected 12 backs of bodies post mortem, furnishes additional information. In several of the bodies a herniation of fat from one layer of fascia to another was found. In 12 biopsies on selected patients suffering from fibrositis of the back, fatty lobules under tension or fatty hernias were discovered. Their removal resulted in cure in most cases or striking relief in those not completely cured. The fat hernia, according to Mylechreest, may be congenital in origin, where a mass of fat passes through one or more layers of fascia through well defined gaps, or acquired, the result of a mechanical or postural factor. The pain of "fibrositis" may be due to pathologic changes found in these areas, namely œdema of the fatty tissue. hæmorrhage, congestion or torsion of the pedicle of the prolapsed lobule. Herz suggests that the operation for the release or removal of prolapsed herniation of fascial fat is not a panacea for all types of low back pain. However, if the possibility of such a lesion is recognized and a careful examination is made for palpable masses which may prove to be trigger points of pain, numerous patients may by this procedure be relieved of a condition that has been incapacitating, sometimes for many years—*J.A.M.A.*, **129**:1264, 1945.

Treatment of Early Syphilis with Penicillin

Leifer (*J.A.M.A.*, **129**:1247, 1945) gives in the following lines the summary of his observations:—

1. Each of 96 patients with early syphilis received sixty intramuscular injections of 20,000 units of penicillin in saline solution at three hours intervals, day and night, for seven and one-half days.

2. A Herxheimer reaction occurred in 88 (91·6 per cent) of the patients, and also some minor cutaneous, gastrointestinal and febrile reactions; in no case was it necessary to suspend or interrupt treatment because of reactions.

3. A relapse was observed in 8 (8·3 per cent) of the 96 patients, but 2 of these may have been reinfection. Relapse occurred in 1 (5·3 per cent) of 19 seronegative primary cases. in none of 34 seropositive primary cases and in 7 (16·3 per cent) of 43 secondary cases.

4. Of 72 patients followed from twelve to twenty months, 70 were seronegative and 1 had a doubtful Kahn test of the blood at the last visit; the other patient relapsed in the thirteenth month, and he may have had reinfection.

5. The cerebrospinal fluid of 89 patients was reexamined six months or later following treatment; it was normal in 86, including 8 with abnormal pretreatment fluids; the 3 abnormal fluids were coincidental with a clinical or serologic relapse three to four months after treatment.

6. Although the results of this study must be considered preliminary—and the optimum treatment schedule, particularly for secondary syphilis, is still to be worked out—the findings suggest that the eight day treatment of early syphilis with 1,200,000 units of penicillin does cure early syphilis.

Recognition of Primary Hyperparathyroidism

Keating and Cook (*J.A.M.A.*, **129**:994 1945) write that twenty-four cases of hyperparathyroidism have been observed at the Mayo Clinic in a period of less than two and a half years. In the preceding fourteen years only 14 cases were encountered. The increased incidence is entirely a reflection of a higher index of suspicion and revision of the criteria necessary to make the diagnosis.

In 7 of these cases there was classic osteitis fibrosa cystica generalisata. In 9 there was mild or atypical bone disease and in 8 there was no evidence of bone disease whatever. Calcification of the kidneys or renal calculi occurred in 22 of the 24 cases, an incidence of 92 per cent. In 18 cases (75 per cent) symptoms produced by renal calculi were the chief clue to the diagnosis.

The diagnosis depends on the demonstration of an increase of calcium and a reduction of inorganic phosphorus in the serum as well as increased excretion of calcium in the urine. These changes may be slight, and prolonged study is frequently required to establish their significance.

On the basis of these data the authors wish to reemphasize the conclusions of Albright and his collegues that (1) hyperparathyroidism is more common than generally supposed, (2) renal involvement is more frequent and diagnostically more important than osseous involvement and (3) every patient who has renal calculi should be suspected of having hyperparathyroidism until the contrary is clearly proved or until some other etiologic factor can be clearly demonstrated.

Sixth Nerve Paralysis After Spinal Analgesia

Fairclough (*Brit. M. J.*, **2**:801. 1945) writes that during the period under review spinal analgesia was administered to 2,021 patients. Ten of these developed paralysis of the sixth

nerve. Though the number of cases is not large it indicates that abducens paralysis may be expected to occur once in every 202 cases after spinal analgesia. Premedication was given in all cases, and in a number of instances the lumbar injection was supplemented by the use of general anæsthetics gas-and-oxygen, pentothal, and others. Of the 10 cases reported 6 were female and 4 were male. The right abducens was affected in eight instances and the left in two. All the patients considered the headaches and double vision the most distressing part of their surgical experience, and some stated that the headache was almost intolerable.

The prognosis is, therefore, ultimately good though occasionally the complications may reactivate existing neuropathic conditions which may prove fatal.

Treatment—Sedative drugs are not very effective in treating the severe basal and general headaches, but keeping the head in the horizontal position gives some relief. The inhalation of oxygen, using the B.L.B. inhaler, has given definite relief in many cases. Galvanism for the paresis is of psychological value only.

For the diplopia, covering the deviating eye gives relief, but continued covering is not advised, as thereby any subconscious effort to fuse images and re-establish binocular vision will not be made. Dark glasses should be worn, with the outer third of the glass before the affected eye made opaque with, say, adhesive black paper. It is probable that muscle exercises and fusion-training may assist in re-establishing parallelism of the eyes and binocular vision in cases in which natural recovery is delayed.

Treatment of Peptic Ulcer with Amino Acids

A recent report by Co Tui and his co-workers (*Gastroenterlogy*, **5**:5, 1945) from several departments of the New York University College of Medicine present some rather astonishing results from the treatment of patients with peptic ulcer by high amino acid feedings. Twelve patients with duodenal ulcer, 5 with combined duodenal and gastric ulcer, 7 with gastric ulcer, I with suspected marginal ulcer and 1 with radiologically confirmed marginal ulcer were placed on feedings at two hour intervals of dextrimaltose and a preparation made by hydrolyzing amino acids. Included in the group were the 4 original patients with peptic ulcer who were given the treatment prior to contemplated gastrectomy. The mixture fed contained from 0·5 to 0·8 Gm. of nitrogen and 40 to 50 calories per kilogram of body weight in twentyfour hours. The length of the ulcer history varied from one month to twenty years; 14 were "intractable" cases, as defined by symptoms which had not improved after complete bed rest and strict Sippy treatment for a period of one to five weeks. There were 27 cases in which pain or epigastric distress was the persistent symptom; this stopped in twenty-four hours in 14 and in forty-eight hours in 23. Vomiting a symptom in 13 cases, stopped immediately after the institution of a diet in 6. Occult blood was present in 15 cases and disappeared within one week. The New York investigators believe that the prompt improvement manifest in these cases appears to be due to the protein hydrolysate, which is simultaneously an antacid and an easily assimilable food which can be built into the tissue. In the course of this work the protein deficiency was demonstrated in large percentage of patients with peptic ulcer. The treatment, they point out, does not insure against recurrences when the patient reverts to old dietary habits. Although the number of cases is small and the period of observation short, this therapeutic and supportive use of high caloric and high amino acid feelings deserves to be studied extensively by other qualified investigators.—*J.A.M.A.*, **129**: 954, 1945.

Prophylaxis of Paralytic Ileus by Administration of Morphine

Chestermann and Sheehan (*Brit. M. J.*, **2**:528, 1945) write that morphine is of value in the prophylaxis of "paralytic ileus". The accepted definition of post-operative "paralytic ileus" is the failure to pass flatus within 60 hours at the end of an operation, and Cæsarean section, owing to the laxity of the abdominal wall and hæmoperitoneum, is recognized as predisposing to this condition.

The data submitted for analysis consist of particulars relating to 500 consecutive patients upon whom Cæsarean section was performed when under the care of Dr. W. J. Clancy in the City General Hospital, Sheffield. In the pre-morphine period the drug was given only when necessary to relieve pain, while in the second period the routine was as follows: Morphine gr. ¼ was given 6-hourly for the first 24 hours (if the patient was asleep the injection was given on awaking, and so on 6-hoursly). If distension was present the morphine was continued in the same way for a second period of 24 hours. In any case morphine gr. ¼ was given at night for the first four nights. Fluids were limited to 3 pints a day until flatus was passed, after which the diet was rapidly increased to normal.

There were 160 Cæsarean sections before the morphine routine was introduced. with 8 cases of ileus, and in the second period there were 340 Caesarean sections, with 3 cases of ileus, thus giving rates of 5% in the pre-morphine period as compared with 0·88% in the morphine period, a difference of 4·12:1·40, which is greater than could easily be ascribed to chance. The statistical problem, therefore, is whether it may be validly deduced that the lower rate could have resulted from the institution of morphine therapy.

Indications for Surgery in Penetrating Wounds of the Chest

Qvist (*Brit. M. J.*, **2**:521, 1945) writes that the prognosis of pentrating wounds is worse than that of non-penetrating injuries because pulmonary re-expansion is slower and pleural infection is more likely.

Pneumothorax is of great significance in penetrating wounds and has an unfavourable influence both in the occurrence and resolution of infection and in the resolution of pulmonary contusion.

Pulmonary contusion and laceration are important causes of delayed expansion of the lung and are nearly always found in the condition of unresolved hæmothorax.

Penetrating wounds of the upper lobe have the worst prognosis and are nearly always present with total or apical unresolved hæmothorax.

After surgical evacuation of hæmothorax complete expansion of the lung may still not occur owing to residual pulmonary damage. Treatment for this condition is unsatisfactory, and the best at present is probably prolonged pleural drainage, preferably with negative pressure. Decortication is probably of value in late cases.

Infection of hæmothorax following penetrating and perforating wounds occurred in less than 14% in 400 cases.

Ocular Signs of Ariboflavinosis

Much confusion still exists concerning the oculur signs of ariboflavinosis, or riboflavin deficiency. After the publication

in 1940 of the observations of Sydenstricker and his colleagues (*J.A.M.A.*, **114**:2437, 1940) on the ocular manifestations of this condition, circumcorneal injection and corneal vascularization were accepted as certain diagnostic signs and used in nutrition surveys as an index of riboflavin nutrition. On the basis of ocular signs the incidence of ariboflavionsis has been reported as high as 99·5% and as low as 3% in the subjects examined. The incidence reported seems to depend on whether the observers were ophthalmologists—and therefore conversant with the appearance of the limbus under different conditions and with the different kinds of corneal vascularization or not. The ophthalmologists report a low incidence of ariboflavinosis based on ocular signs. Bessey and Wolbach (*J. Exper. Med.*, **69**:1, 1939) showed that the earliest detectable sign of ariboflavinosis in rats is corneal vascularization. Since then some observers have extended this to include circumcorneal injection, or engorgement of the limbic loops. Others have not discriminated between this and ciliary congestion, and between an engorged limbus and an actual invasion of the cornea by new vessels.

Is circumcorneal injection pathognomonic of riboflavin deficiency? Stannus (*Brit. M. J.*, **2**:103, 1944) has examined between four thousand and five thousand eyes by slit-lamp microscopy, and has seen both corneal vascularization and circumcorneal injection without any evidence of ariboflavinosis. The vessels of the limbic plexus may appear avascular because they are constricted and empty. The slightest stimulus may cause them to become congested and engorged. This occurs in all varieties of conjunctivitis, in those whose eyes are exposed to heat and dust, cold wind. bright light, and mild infection. It may also result by just rubbing the eye, and may come and go in the same individual with considerable rapidity. Engorgement of the limbic vessels is, therefore, not a sure sign of ariboflavinosis, which should be diagnosed only when new capillaries are observed budding from the apices of the limbal loops and extending on to the cornea. Even then the change should be present in both eyes and round the whole corneal circumference, and it should respond rapidly to treatment with riboflavin. Corneal vascularization may also occur in the absence of ariboflavinosis. Thus it has been noted as a result of vitamin-A deficiency, tryptophane deficiency, injury to the corneal epithelium by chemical irritants, and diseases such as trachoma, phlyctenular keratitis, and any superficial keratitis. The instillation of soap solution into the the conjuctival sac may also cause collapsed afunctional blood vessels in the cornea to become engorged.

Ida Mann (*Am. J. Ophthal.*, **28**:243, 1945) has recently recorded a new type of corneal lesion due to riboflavin deficiency. The cornea shows marginal vascularization, which is distinctive and differs from that hitherto described, is in the form of parallel, radially arranged loops springing from the apices of the loops of the limibic plexus and extending over the cornea in three arcades beyond this, slightly more than half-way to the pupil margin. The vessels, which are so engorged that they stand out from the surface, are mostly subepithelial, although in the region of the opacities they penetrate the substantia propria. They extend all round the circumference of the cornea. This is probably a true manifestation of ariboflavinosis as the engorged vessels rapidly empty and the opacities clear after treatment with riboflavin.—*Brit. M. J.*, **2**:89, 1945.

NOTES & NEWS

LICENTIATE OFFICERS OF THE I.M.S. & I.A.M.C. AND POST-GRADUATE DIPLOMA EXAMINATIONS BY THE TWO ROYAL COLLEGES

The Royal College of Physicians of London and the Council of the Royal College of Surgeons of England have approved a recommendation of the Committee of Management of the Examining Board in England to the effect that officers of the I.M.S. or of the I.A.M.C. who are not graduates in medicine and surgery of a University recognised under the regulations of the Board and who desire to take the diplomas of the Royal Colleges, may be admissible to the several examinations by means of special facilities granted solely on account of their War Service, as under:—

I. *For the diplomas of L.R.C.P., M.R.C.S.*

An officer of the I.M.S. or the I.A.M.C. who produces a personal recommendation from the Director of Medical Services to the effect that he is in every way a suitable candidate, will subject to the production of evidence of his medical qualification be admissible without any further certificates to Part I of the First L.R.C.P., M.R.C.S. examination (anatomy and physiology). When he has passed Part I he will be required to complete twelve months' clinical work in a recognised general hospital either in this country or overseas, and to pass Part II of the First examination (pharmacology and materia medica) before admission to the Final examination.

II. *For any of the postgraduate diplomas granted jointly by the two Royal Colleges*

An Officer of the I.M.S. or of the I.A.M.C. who produces a personal recommendation from the Director of Medical Services to the effect that he is in every way a suitable candidate, will subject to the production of evidence of his medical qualification and on complying with the requirements regarding appointments, courses, etc. laid down in the regulations for the several post-graduate diplomas, be admissible to the examinations.

MEDICAL COUNCIL OF INDIA

The Medical Council of India met for its Twenty-fourth Session at New Delhi on the 23rd March, 1946. The President, Dr. Abraham S. Erulkar, M.D. was in the chair.

The Council elected Dr. K. S. Ray as its Vice-President. Rai Bahadur Dr. B. N. Vyas, General Hay and Colonel Bharucha were elected to be members of the Executive Committee.

Amongst the several important matters discussed by the Council, the question of the establishment of an All-India Medical Register which has been agitating the Indian Medical world for a considerable time was one of the most important. The Council had discussed this question at several of its previous sessions and had made certain recommendations which have been considered by the Provincial Governments as well as by the Provincial Medical Councils. During this Session, the Council gave further consideration to the various points raised by these authorities. As the result of these deliberations, the Council has made certain additions to their previous recommendations, and the All-India Register will now include all persons holding qualifications on the Schedules of the Indian Medical Council Act and those practising modern system of scientific medicine in British India and possess qualifications granted by Licencing Bodies in British India

together with those Indian Nationals who hold foreign qualifications not recognised by the Indian Medical Council recognised and registered by Provincial Medical Councils. These will be divided into three groups and this distinction will remain till such date when there is one minimum standard of medical qualifications established throughout the country. The Council were of the considered view that this was not only a decided advance on the previous position but under the limitations of the existing conditions had the merit of meeting all the various points of view in the country.

The Council also deliberated on the question of the recognition of the medical degree granted by the Agra University. Their Inspectors had already reported on the courses of instructions as well as the various examinations held by the University. The University authorities' observations on the reports of these Inspectors were before the Council. The Council has now appointed two of its members to act as Visitors and to report to the Council as to how far the recommendations by their Inspectors have been carried out.

The question of the consideration of the Bhore Committee Report has been referred to the Executive Committee, a special session of which is being held shortly for the purpose.

The next meeting of the Council has been fixed for the 19th of October, 1946.

POTENTIAL SOURCES OF PENICILLIN

As the result of a systematic survey of the antibiotic-producing capacities of penicillin contained in the National Collection of Type Cultures, which has been carried out at the Indian Institute of Science, Bangalore, 12 strains of penicillia have been selected for further study as potential sources of penicillin. Details of the survey have been published in the December issue of the *Journal of Scientific and Industrial Research.—Indian Information*, **18**:193, 1946.

MILK IN INDIA

In a report Mr. A. A. Pepperall, an officer of the British Milk Marketing Board, who toured this country as Milk Marketing Adviser to the Government of India last year, criticizes the methods by which milk is generally produced and distributed in India.

He says: "The present degree of efficiency of the dairy industry throughout India is probably the lowest in the whole world, and records complete disagreement with the existing arrangements." Milk yields, as he finds, of Indian cows are low though they compare more favourably in terms of butter and fat. The best means of increasing the yield is better management of indigenous stock rather than importation of new blood from abroad as India's cattle have to fulfil the dual purpose of providing draught animals as well as milk supplies.

Referring to the present ways of distribution of milk Mr. Pepperall opines that a survey of squalor, filth, ignorance or indifference towards hygienic principles and revolting personal habits allied to official apathy produces in a Western mind feelings of disgust and possibly of despair at the prospect of effecting any appreciable improvement. "Conditions are bad in Bombay, but are perhaps worse in Calcutta where many thousands of buffaloes, together with a similar number of cows, are permanently maintained in stables. Milk is brought from these stables to maket in open galvanized cans slung at the ends of bamboo poles on the shoulders of coolies. The containers are quite open except that leaves or straw are placed on the top to prevent milk from spilling. The market is crowded with people chewing betel nut, smoking and spitting, and there it is tested by prospective buyers dipping their hands in to judge the amount of fat or added water more dipping of hands occurs when milk is measured after sale.

Mr. Pepperall blames Calcutta Corporation for not treating milk as a highly valuable food and attending to the way it is handled. Hundreds of cans arrive daily at a railway station, "open at the top except for the covering of straw or palm leaves." A test of milk in Bombay showed bacteria count of 36 million per c.c.—a fact that presumably inspired Mr. Pepperall to draw the unfavourable comparison with London's sewage. Tests of 247 samples of milk at Calcutta disclosed added water in every case and few specimens that reached the prescribed minimum for butter and fat were in all probability also adulterated as legal definition of pure milk in India places the fat content about two per cent below the normal average of fat found in milk produced by indigenous animals. At least one-fifth of the fat may, therefore, be abstracted without penalty.

The general average of added water exceeded 25 per cent and in one case was 87 per cent and water is at times taken from stagnant tanks. That the poverty-stricken producer who is totally at the buyer's mercy in respect of price he receives, should seek redress by adding water to increase the milk he has to sell is the "inevitable consequence of a contract entered into in bad faith whereby the producer knows that on the question of payment he is being exploited and the buyer in fixing his price expects (and gets) an adulterated article."—*Calcutta Municipal Gaz.*, **43**:213, 1946.

PROSECUTION UNDER DRUGS CONTROL ORDER QUASHED IN PATNA

The Hony. General Secretary, the Patna Meedical Association (Branch of the Indian Medical Association) reports below a case under Drugs Control Order in which a doctor's compounder was unnecessarily harassed. The doctor appealed to the I.M.A. for help and the Secretary helped him in the case. The report of the case is as follows:—

Khan Shahib S. A. Ashraf, Senior Deputy Magistrate who is also in-charge of Miscellaneous Civil Supplies including drugs went to the Regal Pharmacy, Patna on 24-9-45 and demanded one tin of Glucose 'D' of Glaxo brand. Mr. Monoranjan Paul replied he had no tin of Glucose D (Glaxo) and offered him one tin of India make. Khan Sahib entered the compounding room and found one tin of Glucose D (Glaxo). He seized the two tins and sent them to S.I. of Police with the report. On his report Mr. Monoranjan Paul was prosecuted under the section of rule 81(4) of D. I. Rules with Section 11 of the Drugs Control Order (1943).

Khan Shahib on being cross-examined admitted that the tin seized by him was open and not full and sealed and he did not mention in his report that he recovered one full and sealed tin of Glucose D (Glaxo). It is thus clear that the accused had no sealed tin in shop and it cannot be said that he refused to sell a tin of Glaxo brand.

Mr. Paul was honourably acquitted by the trying judge.

INDIAN CHEMICAL MANUFACTURERS' ASSOCIATION: GOVERNMENT'S QUININE POLICY

The Indian Chemical Manufacturers', 'Association has issued the following statement to the press with regard to the supply position of quinine in the country.'

"Recently a Press Note has been issued by the Government in connection with the production and distribution of quinine. While the Government have taken great pains in explaining their quinine policy, especially with regard to the stock position of quinine in Government factories, the statement has hardly proved convincing. It is most surprising that out of a total production of 115,000 lbs. of quinine, the Government have thought it fit to reserve stocks upto the extent of 75,000 lbs. in spite of the heavy loss of life in the country due to malaria epidemic.

"Such large reserves are absolutely out of proportion to the quinine production in the Government factories and it is but natural that with such large stocks in the factories, there could be no enthusiasm to push up the production of quinine in Government factories. It is not understood as to what made the Government so over-cautious as to reserve such huge stocks out of the quinine produced in Government factories if they were sure of similar or increasing production every year. It is indeed difficult to understand this policy of denying quinine to a malaria-stricken people in the face of such stocks and increased production as claimed by the Government.

"It would be interesting to note in this connection that while foreign importers are now supplying quinine at a prohibitive price in India, the Government is contented in keeping their stocks for some future occasion best known to them. How is it that quinine is now being imported from England at a high price especially when she has no source of quinine supply of her own? This round-about method of supplying quinine to India by giving advantage of middlemen's profits to foreign exporters is no compliment to Government's quinine policy. Since the last several years the Association has been making efforts to obtain a meagre quantity of about 12,000 lbs. of quinine for distribution all over India among chemical manufacturers for the manufacture of quinine ampoules which have been in such short supply during the course of the War. But the Government have refused to make any quinine available out of their stocks on the plea of shortage of quinine. At a time when stocks of quinine have been piling up in Government factories and the Indian market is being captured by imported synthetic anti-malarial drugs, one is led to believe that Government's quinine policy is not helping any one, excepting the foreign interests."

U. P. MEDICAL COUNCIL

The following extracts from the proceedings of the meeting of the United Provinces Medical Council held in its office at Lucknow on 16-3-46 are published for general information:

The case against Dr. Syed Asad Ali, L.M.P., was considered and, after hearing him, his counsel and his witness, Mr. Babu Ram Bajpai, the Council judged him not guilty. The decision was communicated to him by the President.

"Resolved that the United Provinces Government be requested to kindly take steps as to introduce the teaching of 'Elementary knowledge of Public Health and Hygiene' in all the schools of these provinces under the Department of Public Instruction, as these subjects command a strategic position in the preservation and uplift of National Health of a country, and that the Council bring it to the notice of the Government that these subjects, popularly known as 'way to health' were being taught in all the vernacular and high schools of these Provinces in the past but for the reasons best known to the educationists of the said department of Public Instruction were discontinued."

"Resolved that the representative of this Council on the Board of Education be requested to press the medical and health reforms to be introduced in the Education Department such as treatment of boarders, teaching of Hygiene, etc."

MEDICAL PRACTICE IN SIAM

Major Lee, a member of the medical personnel of the I.N.A. in Malaya, Siam and Burma, who has lately been released from the Red Fort, Delhi, and who is at present organising relief to the I.N.A. personnel in U.P. addressed a meeting of the members of the Lucknow Medical Association on the 28th April, 1946. At the outset Major Lee justified the formation of the I.N.A. and referred to its success as being mainly due to the urge for liberating one's own country from foreign domination.

Major Lee who started his medical career in India began to practise in Siam at a time when there was no law restricting the practice of medicine. In 1932 after the revolution, however, a Medical Board was set up which examined all foreign diploma or degree holders before certifying them fit for practice in Siam, knowledge of Siamese being an essential factor. As the number of diploma or degree holders was inadequate at the time, a second class of practitioners was licensed temporarily. This class consisted of those who had some knowledge of medicines. Such practitioners were not allowed to perform operations, administer chloroform, give intravenous injections, use dangerous drugs or visit patients at home and charge fees for such visits. This class was to be statutorily abolished in 5 years when it was expected that the number of the first class of doctors would increase sufficiently.

As in the continent of Europe and most other countries, Major Lee continued, the only basic qualification recognised in Siam was the University degree. Obscene advertisements were prohibited and no patent medicine could be advertised or sold unless the formula was tested and approved by the testing laboratory. The restrictions stated above and the law prohibiting indigenous doctors to use freely medicines and appliances of the modern scientific system were in marked contrast to what obtains in India where anybody can use the word 'Dr' before his name and prescribe the most potent and dangerous drug with impunity. Because of such conditions Indian doctors were looked upon as quacks in Malaya, Siam and other foreign countries of the East and a most rigorous enquiry was instituted before they were proved to be really trained and qualified. Major Lee pointed out that it was high time that the Government of India took steps to put things right. He was certain that if the I.N.A. came to India in the position they intended to they would not have tolerated the prevalance of quackery.

Major Lee gave a comparative picture of the incidence of epidemics in Burma and Malaya during British administration and how within 6 months of Japanese occupation all epidemics were controlled with compulosry inoculations under proper discipline although means of communication, watersupply and drainage, amongst others, were disorganised due to war conditions.

In referring to the post-revolution health reforms in Siam, the speaker narrated how propaganda measures detailing deficiency of the country and requirements necessary by means of the radio and through the press were followed by enactments.

The urgent necessity of rest for medical practitioners which was never enjoyed by doctors in India during their entire professional career was stressed by the speaker. In this connection he dwelt on the advantages of combined practice and how this was carried out in Siam.

CORRESPONDENCE

The Editor is not responsible for any views expressed by the contributors

PROGRESSIVE PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY

SIR,—Will any member or other medico let me know direct or through your columns about the following regarding progressive pseudohypertrophic muscular dystrophy?

1. Latest treatment and monographs with the details of earlier publications with name of author, publisher, price and where available in India.

2. Name and address of any institute or worker in India or abroad carrying on research in this line.

3. Any electrical or surgical therapy tried with success in India or abroad. I am etc.

21-4-46 S. V. VARTAK, L.C.P. & S. (BOM.)
Rewari (Punjab) *Hony. Secretary, I.M.A. Rewari Branch (Punjab)*

CONSULTANTS AND ATTENDING DOCTORS

SIR,—I shall feel obliged if you publish the following in your columns as the points raised affect general practitioners and consultants.

Suppose a medical man attends a case, a consultant or any other medical man is brought in without the knowledge of the attending doctor. He prescribes something for the patient without inquiring of the attending doctor. (1) Is it fair or ethical of the consultant to go without being asked by the attending physician? (2) Is it not his duty to prescribe after inquiring from the attending physician? (3) If the attending doctor as a mark of protest, in presence of the consultant withdraws and refuses his further service for the particular case, is he justified? I am etc.

26-3-46 D. T. ANKLESARIA, M.B.B.S.
Bombay

DISPENSING BY UNREGISTERED PERSONS

SIR,—Despensing by unregistered persons is prohibited in the Pharmacy Bill to be introduced in the Central Legislature.

1. But what about the quacks? The non-qualified compounders of allopathic doctors (and also of quack doctors) are practising allopathy and obtaining degrees of doctors by correspondence from bogus homœopathic medical colleges and thus they are much better off than the qualified compounders.

(Continued in col. 2)

REVIEWS

ANNUAL REPORT OF THE AYURVEDIC DISPENSARY FOR THE YEAR 1938—1939 AND 1939-1940. TRIENNIAL REPORT OF THE AYURVEDIC DISPENSARY FOR THE YEARS 1939-1941. Published by the Secretary C/o. The Charaka Aushadhalaya, Walker Road, Nagpur City, C.P.

The three reports contain the results of treatment on various ailments by Ayurvedic medicines. It appears that favourable results were obtained in many diseases. In each report suggestions have been made to doctors to adopt some of the drugs which have been found helpful.

B. P. NEOGY

MEDICAL ANNUAL, 1935, Edited by Henry Tidy and A. Rendle Short. Royal 8 vo. pp. 410+lxv+75. Published by John Wright & Sons Ltd., Bristol. Price 25 s.

We are glad to tell that the classical annual record of the year's progress has again reached us. In our last review we mentioned that a review on Penicillin would have been timely. It has now appeared. Primary atypical pneumonia, as a new disease, justly occupies a place in its contents. We find that three articles published in our Journal have been included in this review. Work of Dr. P. Sanyal on hernia, of Dr. S. W. Hardikar and V. Gopal Rao (a printing mistake has occurred, Rao has been spelt as Ras) on ascites and of Dr. S. G. Vengsarkar and J. V. Rangnekar on small pox and chemotherapy found their proper places. We again heartily recommend this volume to general practitioners and specialists alike.

N. G. MOJUMDER

(Continued from col. 1)

2. The layman are working as doctors with homœopathic degrees but practising allopathy and homœopathy giving intravenous N.A.B. injection using morphine and penicillin and also performing operations.

3. I think a day will come when the old illiterate servant of a doctor will begin practice because he knows most of the medicines.

4. When will these homœo medical colleges close?

5. Will these existing quack doctors be registered in the same manner as the non-qualified compounders of 3 years' experience before 1942 September are registered in the U.P.? I am etc.

26-3-46 BALWANT RAI AGARWAL.
Ghaziabad

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